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## Original Communications

### THE ELECTRO-ENCEPHALOGRAM IN PATIENTS UNDERGOING OPEN INTRACARDIAC OPERATIONS WITH THE AID OF EXTRACORPOREAL CIRCULATION


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THE electro-encephalogram has been continously monitored throughout operation in all patients undergoing intracardiac operations with the aid of extracorporeal circulation at the Mayo Clinic. The electro-encephalograph was originally included in the monitoring devices in order that specific information on the depth of anesthesia would be available at all times. This desire has been realized. The electro-encephalographic record has provided additional, unexpected information which has been extremely helpful in the management of these patients. This communication is concerned with data from the first 100 patients in whom technically satisfactory electro-encephalograms were obtained.

#### MATERIAL AND METHOD

The electro-encephalogram was obtained through the use of one channel of a 2-channel recording device similar to that developed by Courtin, Bickford, and Faulconer[1] for the study of the effect of depth of anesthesia on the electro-encephalogram. One stainless-steel wire electrode was placed in the scalp 1½ inches from the midline in the frontal region, and one electrode was placed in the ipsilateral occipital region. After suitable grounding had been achieved, the electric energy from these electrodes was amplified and recorded on a continuously moving paper record. The material from these records furnishes the basis for this report.


Read at the Thirty-seventh Annual Meeting of The American Association for Thoracic Surgery at Chicago, Ill., May 4-7, 1957.



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The majority of the patients were children, but several infants as well as several adults were included in the group. Details concerning the operation of the pump-oxygenator[2] and the physiologic variables noted during operation[3] have been reported. The rate of blood flow from the machine to the patient was determined only by the return to the machine from the patient. Flow was neither predetermined nor preset. The averages of the mean rate of flow, classified by body weight of patient, are presented in Table I.

TABLE I. AVERAGES OF MEAN RATE OF BLOOD FLOW

| BODY WEIGHT (KG.) | AVERAGE FLOW (C.C./KG./MIN.) |
|---|---|
| Less than 10 | 89.3 |
| 10-20 | 75.4 |
| 20-30 | 62.2 |
| 30-40 | 58.8 |
| 40-50 | 47.7 |
| More than 50 | 47.8 |

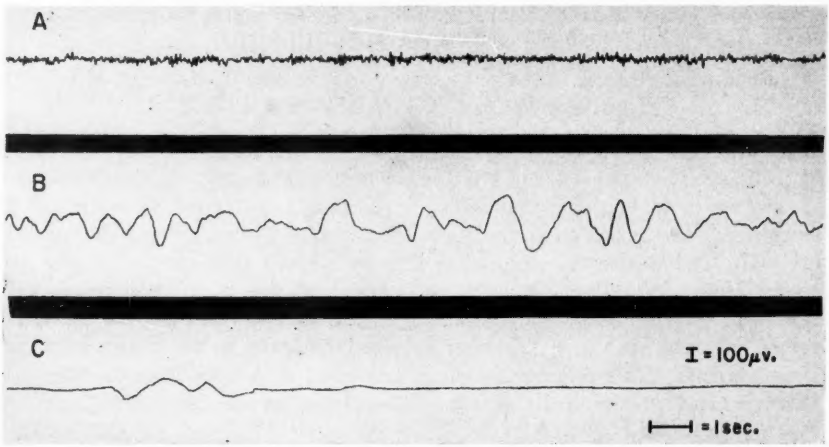


Fig. 1.—An electro-encephalogram characteristic of (*A*) light anesthesia with ether and (*B* and *C*) showing the alteration described in the text.

A steady state of light anesthesia with ether was maintained in all patients. Ether vapor was introduced intermittently into a closed circuit only as necessary to prevent movement of the patient and to help achieve control of ventilation. During the period of perfusion of the patient, a portion of the oxygen delivered to the oxygenating chamber of the apparatus was diverted through an ether vaporizer in amounts sufficient to maintain the light level of anesthesia. In most cases, ether vapor had been introduced into the oxygenator chamber a few minutes before actual perfusion had begun in order to prevent rapid washout of the agent from the patient.

The electro-encephalogram of level I ether anesthesia is characterized by waves having a frequency of 20 to 24 cycles per second and an amplitude of 30

to 40 microvolts[4] (Fig. 1). The pattern of waves of low amplitude and high frequency, characteristic of light ether anesthesia, will be referred to as the "normal electro-encephalogram."

Alterations in the electro-encephalogram due to increased depth of anesthesia have been reported.[1] They are easily recognized, being gradual in onset and associated with addition of ether vapor to the respiratory circuit. This alteration will not be considered in this report.

## RESULTS

A normal electro-encephalogram predominated in all patients before, during and after perfusion. However, electro-encephalographic alterations not associated with variations in anesthetic depth were observed. The alterations consisted of disappearance of waves of low amplitude and high frequency and replacement of them by waves having a frequency of 1 to 2 cycles per second and an amplitude of 100 to 300 microvolts. This electro-encephalographic variation was observed 71 times. On some occasions these slow, large waves gradually disappeared and the electro-encephalogram became flat (see Fig. 1).

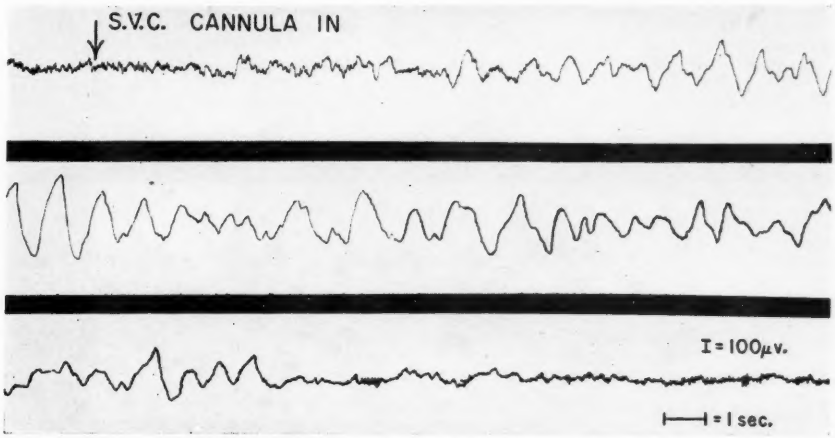


Fig. 2.—A continuous electro-encephalogram demonstrating the transition from normal to abnormal pattern with temporary obstruction of the superior vena cava during insertion of a cannula. The normal pattern reappeared after the cannula was in place.

On 13 occasions the variation followed cannulation or surgical compression of the superior vena cava. In each instance the normal pattern returned with resumption of flow through the superior vena cava. An example of such an incident is shown in Fig. 2.

The electro-encephalographic change was associated 12 times with a reduction of the patient's cardiac output and once with an interruption of perfusion. Descriptions of these 13 instances, with the treatment and result, are given in Table II. Identical incidents in the same patient are tabulated as one occurrence. Examples from this group are depicted in Figs. 3 and 4.

TABLE II. INSTANCES OF ASSUMED OR KNOWN REDUCTION OF BLOOD FLOW ASSOCIATED WITH CHANGE IN ELECTRO-ENCEPHALOGRAM

| DESCRIPTION OF INCIDENT | TREATMENT | RESULTS |
|---|---|---|
| Arterial line of bypass accidentally uncoupled; 6 minutes without flow; patient's temperature 34° to 32° C.; EEG immediately flat | Re-established perfusion | Flat EEG for 21 minutes, followed by large, slow waves for 2 to 3 hours; gradual appearance of fast activity; patient awake and rational with removal of endotracheal tube; uncomplicated convalescence without neurologic defect |
| Gradual circulatory deterioration with severe hypotension (40/30) in periods of 3 to 4 minutes before perfusion | Established perfusion | Reappearance of normal EEG |
| Hypovolemia in the same patient with additional loss of blood on removal of subclavian cannula; onset of flat EEG | Infusion of blood; intracardiac epinephrine and calcium gluconate; cardiac compression | Death |
| Severe hypotension during chest closure (30/25) | Intravenous infusion of Levophed | Normal blood pressure and gradual reappearance of normal EEG |
| Hypovolemia after perfusion; infusion of citrated blood in subclavian artery leading to ventricular fibrillation | Reinstitution of perfusion; defibrillation | Reappearance of normal EEG |
| Complete A-V dissociation after repair of ventricular septal defect; heart rate 24/min.; low mean blood pressure (70 to 90/10 to 20); onset of flat EEG | Intravenous infusion of epinephrine, Levophed, and molar sodium lactate; pacemaker | Death |
| Asystole at cessation of perfusion | Re-established perfusion; intracardiac epinephrine and calcium gluconate | Immediate reappearance of normal EEG; successful cessation of perfusion with normal EEG |
| In the same patient episodes of profound hypotension during closure of chest (40/30) | Intravenous infusion of Levophed | Adequate blood pressure and reappearance of normal EEG (see Fig. 3) |
| Hypotension during chest closure (50/20) | Intravenous infusion of Levophed | Blood pressure increased to 80/40; gradual reappearance of normal EEG |
| Several episodes of profound hypotension after perfusion | Massive doses of Levophed intravenously | Increase in blood pressure and reappearance of normal EEG in early episodes; death finally ensued |
| Inadequate circulation with cessation of perfusion following repair of ventricular septal defect in patient with associated irreparable mitral insufficiency; EEG flat | Re-established perfusion | Immediate reappearance of normal EEG (Fig. 4); perfusion finally discontinued in spite of inadequate circulatory dynamics; death ensued |
| Hypotension with manipulation of heart and great vessels | Cessation of manipulation | Gradual reappearance of normal EEG |
| Terminal circulatory deterioration following perfusion and repair of single ventricle; flat EEG | Intracardiac epinephrine and calcium gluconate; intravenous Levophed | Death |

The electro-encephalographic change occurred 45 times with the onset of perfusion. The duration of the change ranged from 10 to 420 seconds with a mean of 141 seconds. In all these cases the normal pattern spontaneously reappeared and continued throughout the perfusion. Fig. 5 presents an example of this change.

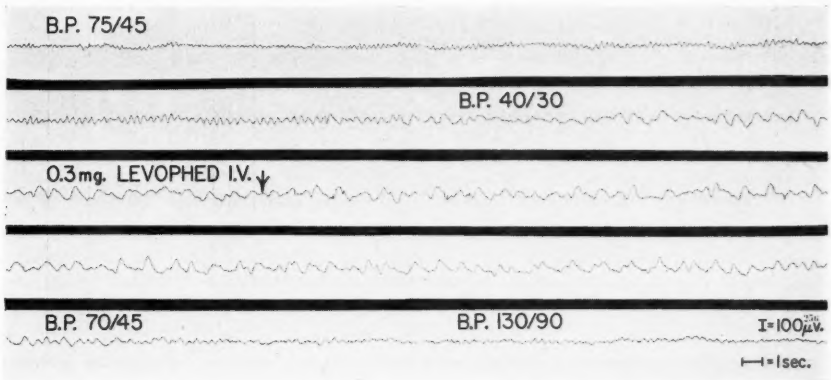


Fig. 3.—Electro-encephalographic changes associated with profound hypotension.

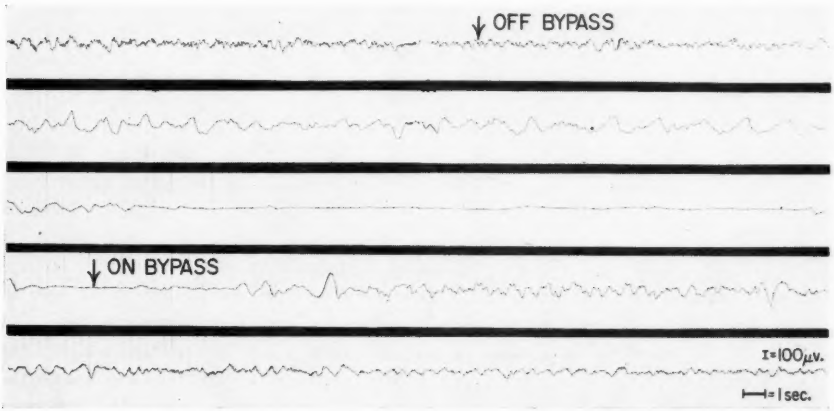


Fig. 4.—A continuous electro-encephalogram showing the alteration with cessation of perfusion in a patient unable to establish normal circulatory dynamics. The normal pattern returns with reinstitution of perfusion.

In these 100 patients the perfusion per se was characterized by a normal electro-encephalogram except for the one instance of interruption of flow due to a technical error.

No patient in this series exhibited postoperative evidence of damage to the central nervous system which could be attributed to the perfusion. Five patients died in the operating room owing to obviously abnormal cardiovascular dynamics and were not evaluated during the postoperative period. One patient

was hemiplegic and died. Necropsy revealed an Ivalon sponge cerebral embolism. One patient awoke from anesthesia and recognized her mother but remained lethargic and died. The venous pressure of this patient was extremely high during the perfusion, owing to serious and unrecognized systemic venous anomalies. The remaining 93 patients showed no clinical evidence of cerebral damage in the postoperative period.

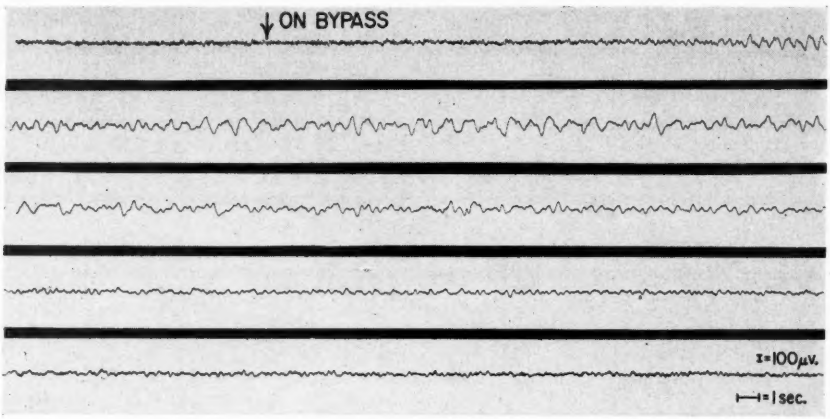


Fig. 5.—An electro-encephalographic change with the onset of perfusion. Spontaneous return to normal takes place.

## COMMENT

Adequate data are not available to correlate, in quantitative terms, cerebral blood flow and the electro-encephalogram. Beecher and associates,[5] in 1938, reported a flattening of the electro-encephalogram in cats, the blood pressure of which had been greatly reduced by induced cardiac tamponade. Lennox, Gibbs, and Gibbs[6] reported similar electro-encephalographic changes in human beings suffering from orthostatic hypotension and sensitive carotid sinus reflexes. Clowes and associates[7] presented electro-encephalographic records from dogs in which perfusions were marked by low blood pressures and low flows and which were in a class of nonsurvivors. The electro-encephalograms of these animals were abnormal, and Clowes observed that survival was associated with a normal electro-encephalogram during the perfusion. No data have been presented on the degree of reduction of total blood flow or cerebral blood flow necessary to produce a change in the electro-encephalogram. No data are available which allow one to derive the cerebral blood flow from the total systemic flow. It is perfectly valid to expect a flat electro-encephalogram during a period of absent total systemic or cerebral blood flow. A flat electro-encephalogram does not, however, necessarily indicate absence of cerebral blood flow. It is not possible to predict the degree of reduction of cerebral blood flow necessary to produce a change in the electro-encephalogram. Within these limitations the electro-encephalogram provides useful information on the adequacy of cerebral blood flow.

Factors other than depth of anesthesia and cerebral blood flow may influence the electro-encephalogram. Hypoglycemia, hypoxemia, hypocapnia, and hypothermia may increase the tendency of the electro-encephalogram to flatten. Lennox, Gibbs, and Gibbs[6] have stated that an electro-encephalographic change appears at a blood sugar level in the internal jugular vein of 50 mg. per 100 c.c. or at a hemoglobin saturation in the internal jugular vein of 30 per cent. Libet and associates[8] reported a reduction of electric frequency and amplitude with decreasing body temperature. At 20° to 23° C., trains of large, slow waves began to appear and these gradually decreased to a flat line at 15° C.

These observations aid in the interpretation of the electro-encephalographic variations found in this study. The variation associated with a reduction in superior vena caval flow may reflect a decrease in cerebral blood flow proportionate to the decrease in total cardiac output occasioned by impaired venous return, or may represent cerebral anoxia incident to stagnation of blood flow because of increased venous resistance. It is probable that both factors contribute to the observation.

The electro-encephalogram has had its most useful application in our interpretation of the degree to which circulatory efficiency has deteriorated. We believe that the manifestations of failing circulation accompanied by an electro-encephalographic change indicate a decrease in cerebral blood flow below a safe level, and require immediate and vigorous treatment. Continued electro-encephalographic normalcy in the presence of altered hemodynamics is a reassurance that serious reductions in cerebral blood flow have not occurred.

The transient abnormality in the electro-encephalogram appearing in some patients at the onset of perfusion is puzzling. Oxygen saturation of the blood returned to the patient has invariably been 100 per cent. Carbon dioxide tension of this blood has not been measured. The variation has appeared both in the presence and in the absence of ether in the perfusing blood. During the transitory period of establishing total perfusion the flow is variable and cerebral blood flow may be inadequate. Dr. W. G. Bigelow[9] has suggested our present working hypothesis, which is that the electro-encephalographic change is occasioned by perfusing the brain with blood which is initially at or near room temperature. As this blood mixes with that of the patient, equilibrium takes place at a temperature between that of the room and the patient. At this time the electro-encephalogram returns to its normal pattern. Studies of the temperature of blood in the arterial line during the perfusion of a subsequent group of patients have supported this hypothesis.

## SUMMARY

1. The electro-encephalograms of 100 patients undergoing open intracardiac operations with the aid of extracorporeal circulation at the Mayo Clinic have been reviewed.

2. The electro-encephalographic pattern characteristic of light ether anesthesia predominated in all patients before, during, and after perfusion.

3. Alterations in the electro-encephalogram not associated with increased depth of anesthesia occasionally occurred with known or presumed reductions in cerebral blood flow.

4. A transient alteration in the electro-encephalogram frequently occurred with the onset of perfusion and was probably based on the lowered temperature of blood in the arterial line.

5. The electro-encephalogram is a useful monitoring device.

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## Discussion

DR. GEORGE H. A. CLOWES, Cleveland, Ohio.—I cannot let such a nice paper pass without comment. I think that perhaps Dr. Theye misinterpreted the paper he referred to. As a result of studies on hypercapnia, hypoxia, depth of anesthesia, following the work of Faulconer and Bickford at the Mayo Clinic, and others, we have come to the conclusion that a flattened brain wave, provided the blood flow is maintained, represents an increase in the depth of narcosis or depression of brain activity. For example, if a dog is given $CO_2$ and the brain wave potential is absent for periods of 15 minutes or more, one can predict that this dog will not recover. He may appear to recover but he will die in a state of shock within the next 24 hours. This is a pretty important thing. It offers some explanation of why dogs and patients who have gone through this sort of thing just seem to peter out later on. They die of hypotension in spite of all our efforts to take care of them. It is probable that emboli and other causes for impairment of circulation to the brain may result in electro-encephalographic depression and ultimate death. I think that this excellent presentation and its careful analysis is a very real contribution.

DR. KARL E. KARLSON, Brooklyn, N. Y.—I should also like to congratulate Dr. Theye and the group from the Mayo Clinic on this very excellent presentation of their electro-encephalographic findings. I would like to comment briefly on the significance of the electro-encephalographic findings particularly as they pertain to cerebral-arterial gas embolism.

(slide) This slide shows representative segments of a 6 lead electro-encephalographic tracing taken continuously before, during, and after the injection of 0.25 c.c. of air per minute

for one hour into the internal carotid artery of a dog anesthetized with Nembutal. The top 6 lines on the slide show the pre-injection electro-encephalogram. The middle 6 lines are representative of the tracing during injection of air. The lower 6 lines show the electro-encephalographic pattern after the completion of injection of the entire 15 c.c. of air into the internal carotid artery. All 3 of the tracings are normal. There was no significant change in the electro-encephalographic pattern during the entire experiment.

This dog did not recover consciousness and subsequently died of cerebral air embolism. These findings would indicate that the electro-encephalogram may not be an accurate indicator of the existence of gas embolism or the extent of brain damage that might occur as a result of cerebral-arterial gas embolism during perfusion with an extracorporeal pump-oxygenator.

# INTRAPERICARDIAL BRONCHOGENIC CYSTS

## Report of Two Cases and Probable Embryologic Explanation

C. Harwell Dabbs, M.D.* (by invitation), Knoxville, Tenn., Ralph Berg, Jr., M.D., Spokane, Wash., and E. Converse Peirce II, M.D.* (by invitation), Knoxville, Tenn.

CYSTIC tumors of bronchial origin are known to occur in many different locations within the thorax. Aside from the usual paratracheal, carinal, hilar or paraesophageal location, they have been reported in association with vertebral bodies,[1] in the presternal area,[2] and intrapericardially.[3, 4] We shall report 2 additional cases of intrapericardial, bronchogenic cysts successfully removed.

### CASE REPORTS

Case 1.—L. M. B., a 42-year-old white widowed woman, developed chills, fever, malaise and a productive cough on Nov. 25, 1954. She was treated with antibiotics and apparently made a good recovery, but after 2 weeks of therapy, fluoroscopy showed nonclearing of the right lung. A roentgenogram showed a shadow in the anterior mediastinum projecting into the right chest and containing cystic calcific densities (Fig. 1). The past history included bilateral congenital cataracts, one operated on successfully 14 years previously. At age 13 she apparently had had a bout of rheumatic fever and had been out of school for one year. She had received digitalis in drop form for one year and, in general, had done well. At age 27, she had a pregnancy terminate in a still birth at 8 months. Apparently she went into cardiac failure at that time. She denied any symptoms suggesting embolization, fibrillation, or other arrhythmia. She occasionally had ankle edema, dyspnea, and irregular heart action on exertion.

Physical examination revealed an obvious right congenital cataract and a deformity of the left iris from cataract removal. Examination of the chest was essentially negative. The heart was clear to percussion and auscultation except for a short, Grade 1, systolic murmur transmitted toward the shoulder and heard best in the pulmonic area. No abnormalities to percussion or auscultation were noted over either lung field. Examination of the abdomen was negative. Fluoroscopy revealed a mass coming from the right hilum which, on the oblique and lateral positions, was found to be anterior. The pulsation of the mass appeared to be transmitted. Barium by mouth filled the esophagus and was not deviated, since it was posterior to the mass, and did not outline any enlargement of the left atrium.

The patient was admitted to the Deaconess Hospital in Spokane, Wash., where routine laboratory data were within normal limits. No special diagnostic studies were carried out, it being assumed that the patient had an anterior mediastinal tumor, probably a teratoma.

Operation was performed on Feb. 17, 1955, the right chest being entered through the bed of the resected fifth rib. The mediastinal tumor was obvious and located anterior to the phrenic nerve occupying the whole right cardiac presenting surface subpericardially. The pericardium was resected around the tumor and the usual free intrapericardial space

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was not encountered. There were numerous adhesions between the heart and all pericardial surfaces. After careful and laborious dissection, a very intimate attachment to the right auricular tip was encountered which necessitated its resection with the invading tumor. Further dissection revealed a broad base of aortic attachment measuring approximately 4 by 2.5 cm. Any attempt to dissect this from the aorta was met with increasing hemorrhage. It was obvious that the blood supply to this lesion was coming from the ascending aorta, presumably due (at that time) to its invasion of that structure. Accordingly the tumor was bisected and it was removed in halves with suture ligatures at the base of the aorta. However, it was very definite that a small portion of an epithelial-lined cystic structure was present in the base which was not removed. This was treated with 7½ per cent iodide and the entire base was outlined with silver clips to facilitate radiation

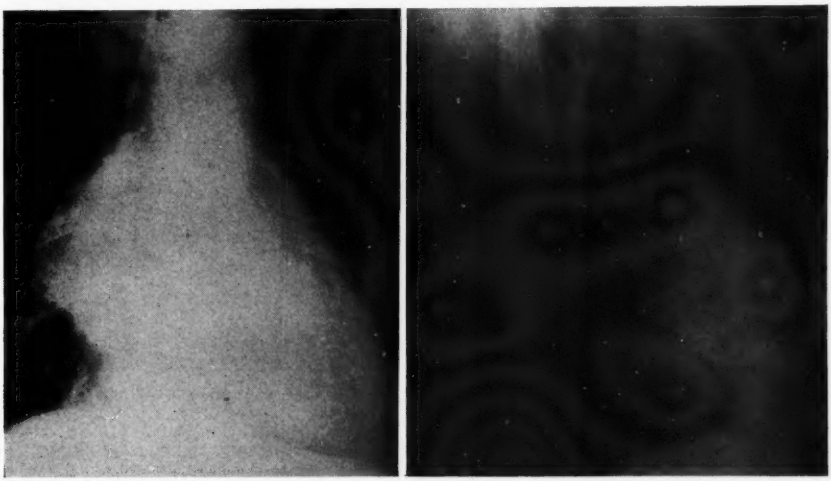


Fig. 1.—Case 1. Roentgenograms of the chest revealed a large mass, partially calcified projecting to the right and anteriorly.

therapy in the future should such a procedure be necessary. The pericardium was closed as well as possible to prevent cardiac herniation. The chest was drained with a single tube and the wound closed in the usual manner.

Postoperatively, the patient had an uneventful course and has remained in good health since surgery. The postoperative chest roentgenogram has returned to normal appearance.

The pathologic diagnosis of miltilocular bronchogenic cyst was made by Dr. John McCarthy.*

CASE 2.—M. L. D., a 20-year-old woman, was referred to the Acuff Clinic on Dec. 30, 1955, for evaluation of a mediastinal mass. She had been in excellent health and had applied for a job in Detroit, Mich., about 3 months previously. A large shadow adjacent to the heart was found on routine x-ray study of the chest, and she was referred to us for further evaluation.

The past history was noncontributory. A normal baby had been born 8 months previously, and there had been no complications either during pregnancy or at parturition. There was no dyspnea, edema, chest pain, cough, fatigue, or other symptoms referable to the cardiorespiratory system.

*Pathologist to Deaconess Hospital, Spokane, Wash.

Physical examination showed a slender woman with full neck veins and a slight prominence of the chest to the right of the sternum. Over the apex of the heart, a blowing systolic murmur was present. The abdomen was soft; the liver was not palpable; the abdominal aorta pulsated normally. The blood pressure in both right and left arms was 120/70 mm. Hg, and the systolic pressure by palpation in the right leg was 110. The venous pressure in the right arm was equivalent to 180 cm. of saline solution (above the right atrium), and that in the right leg to 60 cm. of saline solution (above the right atrium, in the recumbent position). Urinalysis, hemoglobin, total white cell count and differential count were within

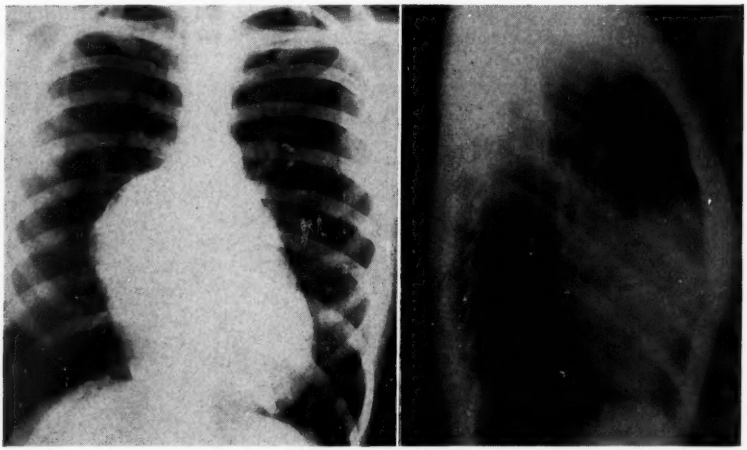
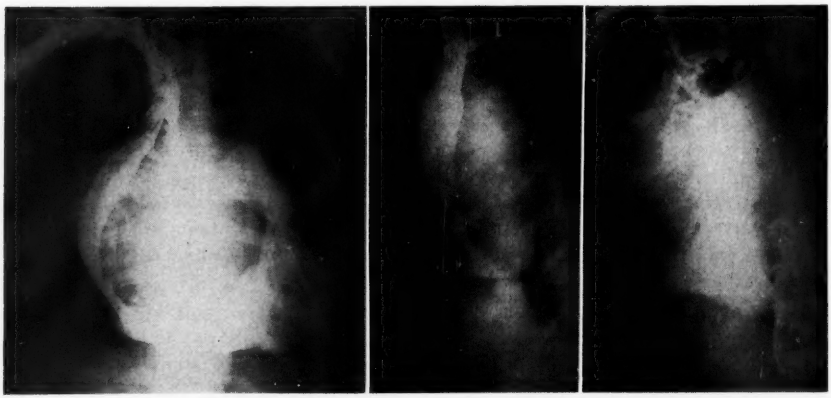


Fig. 2.—Case 2. Roentgenograms of the chest showed identical distortion of cardiac silhouette and calcification within the mass as in Case 1.



*A.* *B.* *C.*

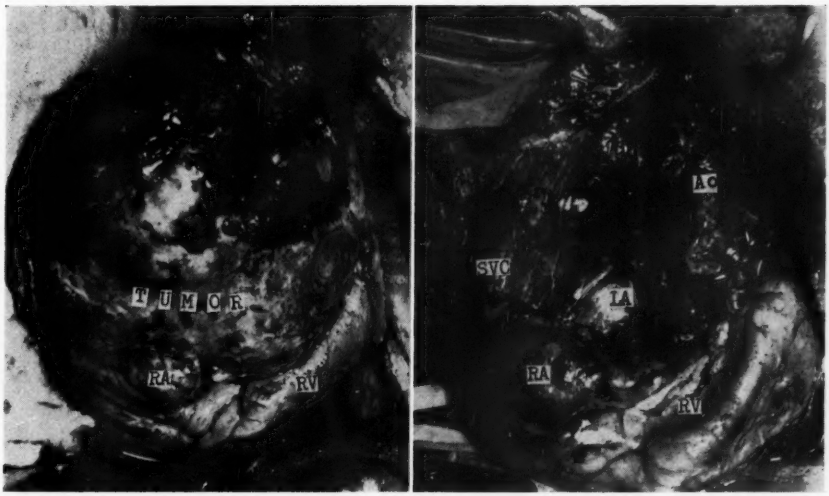
Fig. 3.—Angiocardiograms from Case 2. *A,* The anteroposterior view shows displacement of the superior vena cava to the right and of the right atrium inferiorly and the right ventricle inferiorly and to the left.

*B,* The right anterior oblique shows displacement of the right atrium inferiorly. Note also retrograde filling of the azygos and inferior vena cava, indicating partial obstruction of the superior vena cava.

*C,* The right anterior oblique (later in the series) shows the right atrium lying on the diaphragm. Again note azygos and hemiazygos filling.

normal limits. An electrocardiogram showed a rate of 75 per minute, the P-R interval 0.20 second, and the QRS duration 0.06 second. The P waves in Leads II, III and AVF were biphasic. The tracing was considered normal.

Roentgenograms of the chest showed a very peculiar configuration of the heart with considerable fullness of the pulmonary-artery complex and a rather prominent bulging along




*A.* *B.*

Fig. 4.—Case 2. *A*, Appearance of the tumor at thoracotomy before dissection away from the heart. The position of the heart is the same as in Fig. 3, *A*. The pericardium has been opened widely. Note the right atrium and right ventricle flattened out against the diaphragm. No other portion of the heart is visible.

*B*, Appearance of the heart after removal of the tumor. Note the large concavity, with the superior vena cava entering the right atrium just above the diaphragm and the concave depression in the right ventricle. The left atrium is visible in the depths of the cavity and the root of the aorta is completely "dissected out" by the tumor.

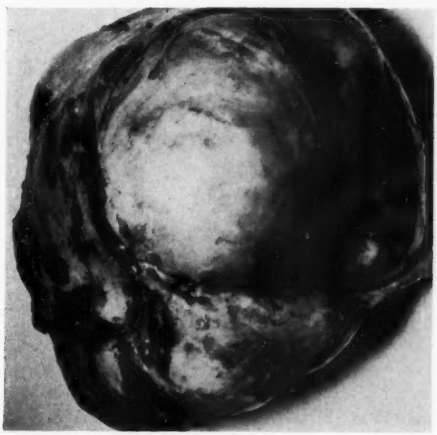


Fig. 5.—Case 2. The tumor before opening weighed approximately 650 Gm. and was about the same size as the heart.

A. B. C.

Fig. 6—Case 2. Comparison of posteroanterior angiocardiogram (*A*), line drawing of angiocardiogram (*B*), and appearance of heart after tumor removed (*C*) illustrates the nicety with which the diagnosis can be made. See Figs. 3 and 4 also.

the right superior border (Fig. 2). The apex of the heart appeared to be somewhat elevated away from the diaphragmatic leaf, suggesting hypertrophy of the right ventricle. There was a peculiar lamellated calcification along the superior portion of the right border, strongly suggesting a cystlike structure. This was seen to be situated well anteriorly in the right lateral view. Barium by mouth revealed displacement of the esophagus somewhat to the left in the posteroanterior view and, in the oblique views, an indentation that was probably due to the calcified mass. Laminagraphic study through the cardiac silhouette showed the mass to be situated well anteriorly, revealing a discrete periphery of calcification and mottled calcification within the confines of the mass. There was no definite "layering" pathognomonic of an echinococcus cyst nor any organized structures suggestive of teratoma.

Fluoroscopy showed a large, calcified mass, 10 cm. in diameter, that was anterior and to the right of the heart shadow but could not be separated from it. The esophagus was deviated posteriorly in both anterior oblique views. The heart appeared normal otherwise. The mass was not thought to be the left atrium because, despite its size, it did not encroach upon the left border of the heart in any location and was too anterior in position.

Angiocardiograms demonstrated a large filling defect in the cardiac silhouette, with marked distortion of the heart (Fig. 3). The superior vena cava was displaced to the right and posteriorly. The right atrium and ventricle were displaced inferiorly and posteriorly. The left side of the heart was displaced posteriorly and far to the left. The resulting defect measured about 10 cm. in diameter.

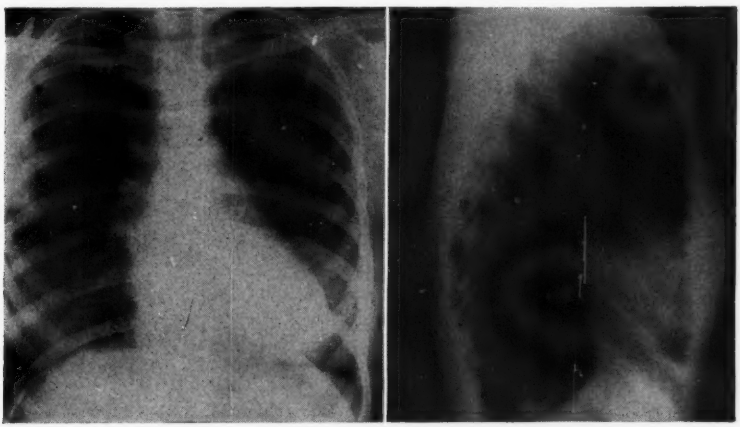


Fig. 7.—Case 2. Roentgenograms of the chest, taken 3 weeks postoperatively, show a normal cardiac silhouette.

It was the consensus of the Acuff Clinic staff that the patient had a tumor, probably within the pericardium and, in view of the great size of the tumor with no symptoms, probably benign. In view of the fact that the tumor was calcified, the most likely diagnosis appeared to be teratoma although the possibility of echinococcus cyst could not be ruled out. The venous pressure, which was elevated in the upper extremity and normal in the lower, led us to believe that the tumor must be intrapericardial, producing obstruction to the superior vena cava, but not intracavitary, for this should have produced obstruction to the inferior vena cava as well. The peculiar manner in which the 2 atria were separated on the angiocardiogram, with the vena cava and right atrium rotated to the right and inferiorly, suggested that the tumor had arisen near the root of the aorta and "dissected" the 2 atria apart as it grew. A search of the available literature at that time disclosed

5 previously reported cases of "intrapericardial teratoma," and we thought that this was the most likely diagnosis. At that time we were not aware of the intrapericardial bronchogenic cyst diagnosed and successfully operated upon by Fontaine, nor were we aware of the unreported cases of intrapericardial bronchogenic cysts of Miscall or of Berg, both of which were successfully removed.

At operation, performed on Jan. 11, 1956, at the East Tennessee Baptist Hospital, with the patient supine, the right side of the chest was entered through an intercostal incision in the fourth interspace. The pleural cavity was free, there was no fluid, and

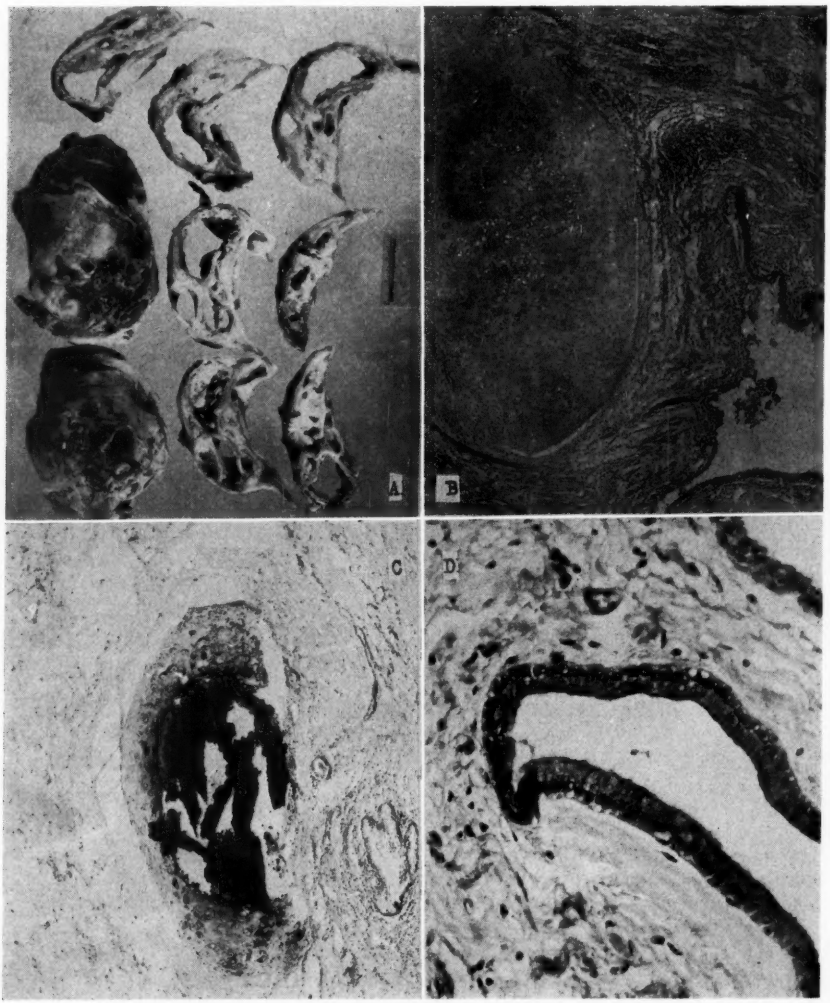



Fig. 8.—Gross (*A*) and microscopic sections of the tumor typifying both cases (kindly supplied by the Armed Forces Institute of Pathology, courtesy Dr. William F. Manion, Chief, Cardiovascular Pathology). Note: (*B*) (×36) from Case 2, the cartilaginous plate, fibrous stroma, cystic spaces lined with bronchial and pseudostratified squamous epithelium; (*C*) (×36), focal calcification; (*D*) (×260), from Case 1, characteristic respiratory epithelium. (All photomicrographs reduced ⅙.)

the lung appeared normal. A firm, large tumor projected anteriorly against the right wall of the chest and the posterior aspect of the sternum, and appeared to lie entirely within the pericardium. It was a large tumor, measuring approximately 10 to 12 cm. in diameter, and was roughly about the size of the patient's heart. It displaced the superior vena cava to the right and inferiorly, and the right atrium and right atrial appendage were markedly displaced inferiorly (Fig. 4).

As the dissection proceeded it was obvious that we would have to have more room, and a transverse sternotomy was performed, carrying the incision into the fourth interspace of the left side and opening the left pleural cavity as well. After this exposure was obtained, it was seen that most of the heart was displaced posteriorly and to the left, as had been demonstrated on the angiocardiograms, with the displacement of the superior vena cava to the right. Two small ateries, arising from the ascending aorta and passing directly into the tumor, were encountered and controlled with suture ligatures. When finally delivered, the tumor consisted of a partially calcified, 650-gram mass, 10 cm. in diameter.

The appearance of the heart after the removal of the tumor was remarkable and was exactly as had been visualized from the angiocardiographic studies (Fig. 4, *B*). The inferior vena cava, which had been plastered onto the surface of the tumor and stretched out over it, was markedly lengthened. It had entered the right atrium at about the level of the diaphragm from a right lateral position. The right atrium lay transversely, flattened against the diaphragm, and the atrial appendage projected directly laterally. The right ventricle and pulmonary-outflow tract were considerably displaced posteriorly and to the left as well as rotated on their axis so that they pointed at about the left axilla, and the left ventricle was indented and displaced inferiorly and to the left. In the depths of this large cavity, which had been dissected among the chambers of the heart by the tumor, lay the left atrium, its anterior wall projecting against what had been the posterior aspect of the tumor and not in apposition with the right atrium at all. In other words, this tumor had apparently arisen between the root of the aorta and the vena cava and had gradually dissected the 2 atria apart, so that the right atrium was markedly displaced inferiorly and to the right. The pericardium was closed loosely with interrupted sutures. An intercostal drainage tube was placed in each chest cavity, and the wound was then closed in layers. The patient tolerated the procedure well.

On pathologic examination, the specimen consisted of a reddish-brown, fluctuant, lobulated mass measuring 12 by 8 by 7 cm. (Fig. 5). Gross section revealed a multicystic architecture, with calcified cyst walls and necrotic, fibrous, intervening tissue (see Fig. 8, *A*). Most of the cysts were filled with a yellow, mucinous, muddy fluid. One cyst contained a white, mucoid substance.

Microscopic examination showed the larger cyst to be lined by a nonspecific fibrous membrane (Fig. 8, *B*). There was extensive fibrosis, inflammation, and necrosis throughout the specimen. Granulomatous reaction, with large aggregates of macrophages, was prominent. There were foreign-body giant cells containing cholesterol clefts. The most common organized structures consisted of respiratory mucosa surrounded by lymphatic tissue. Some of the smaller cysts were lined by squamous epithelium, and others by nonspecific columnar epithelium, unlike the colonic type. Small mucous glands were distributed throughout the tissues in association with the respiratory epithelium. There was no evidence of malignant change. Cartilaginous plates were in evidence at intervals throughout many of the sections. A diagnosis of intrapericardial teratoma was made by Dr. R. J. Leffler.*

After an uneventful postoperative course the patient was discharged on the seventh postoperative day, and remains in excellent health (Fig. 7).

## DISCUSSION

The material from both of our cases was studied extensively by Dr. William Manion, Chief, Cardiovascular Pathology at the Armed Forces Institute

*Pathologist, East Tennessee Baptist Hospital.

TABLE I. SUMMARY OF KNOWN CASES OF INTRAPERICARDIAL

| AUTHOR-DATE AGE-SEX-HOW STUDIED | CLINICAL FINDINGS | X-RAY | SPECIAL STUDIES | SURGERY | PATHOLOGY, GROSS AND MICROSCOPIC | LOCATION-SIZE |
|---|---|---|---|---|---|---|
| Jöel,[7] 1890, 14 yr., M, autopsy | Died suddenly while running for streetcar | No | No | No | Identical with ours | Sitting on pulm. artery, "egg sized" |
| Mouat,[25] 1909, 17 yr., M, autopsy | Sup. vena cava syndrome | No | No | No | Contained hair | |
| Baciegalupo,[26] 1918, autopsy | Not given | No | No | No | Identical with ours | 30 Gm., loc-lft. side of heart betw. aorta and pulm. artery |
| Mathias,[27] 1923, 51 yr., F, autopsy | Died suddenly after operation | ? | ? | Not for tumor | Identical with ours | Rt. side of heart, as big as heart |
| Nossen,[28] 1925, 51 yr., F, autopsy | Died suddenly after surgery | ? | ? | Not for tumor | Identical with ours | Rt. side of heart, as big as heart |
| Grimm,[16] 1927, 3 mo., F, autopsy | None given | Cardiac shadow filled chest | No | No | GI and resp. epithel., cart., hair follicle | Assoc. advent. gr. vessels, sev. times as large as heart |
| Lenkeit,[13] 1928, 21 yr., M, autopsy | Purulent peritonitis | ? | ? | No | Hair grossly | 2 cysts, size of "chesnut" and "apple" |
| Jellen and Fisher,[11] 1936, 3 wk., F, autopsy | Dyspnea, cyanosis, stridor | Rt. chest opaque | Fluoro. | No | Identical with ours | 5 times size of heart |
| Somolinos,[14] 1936, 27 yr., M, autopsy | Attempted drainage; septicemia | Mass left chest | Aspiration | Drainage | Hair and bone grossly, resp. epithel. and cart. | Lft. side, big as apple |
| Meltzer,[29] 1941, 1 mo., M, autopsy | Cyanosis, dyspnea | No | No | No | Identical with ours | Not given |
| Beck,[15] 1942, 22 yr., M, operation | Fever, cardiac tamponade | Heart enlarged to rt. and ant.; calcification | No | Yes 2-stage | Hair foll., GI and resp. epithel. | Rt. side of heart, about size of heart |
| Gebauer,[30] 1943 12 yr, F, autopsy | Dyspnea, cyanosis | Cardiac enlarged tremendous, calcification | No | Yes, died | Squamous epith. glands, muscle, cart. | Loc. same as ours, 15 × 11 × 7 cm. |
| Laipply,[42] 1945, 45 yr., M, autopsy | Not given | Not given | No | No | Identical with ours | Left of aorta, 6 cm. in diameter |
| Willis,[17] 1946, 10 wk., M, autopsy | Choking, dysphagia | Greatly enlarged cardiac shadow | ECG normal | No | Same as ours plus GI epith. | Loc. same as ours, 6.5 × 6.5 × 6 cm. |
| Claireaux,[18] 1951, newborn, F, autopsy | Cyanosis and edema | No | No | No | Same as ours plus GI epith. | Left side, 4.4 × 3.1 × 20 cm., heart 2.5 cm. |
| Bednar,[31] 1950, 2 yr., M, autopsy | Cyanosis and "pneumonia" | ? | No | No | Angioreticulo tissue, cysts lined with cylindrical epithelium | Loc. not clear, 7 × 6 × 5 cm. |

*Most of these were reviewed from the original article. When these were not available or when translation difficulties necessitated omission of data, a blank is shown.

†These appear to be the same case.

TERATOMAS AND BRONCHOGENIC CYSTS*

| BLOOD SUPPLY | RESULTS OF AUTOPSY OR SURGERY | BENIGN OR MALIGNANT | ASSOCIATED CONGENITAL DEFECTS | COMMENTS | BRONCHOGENIC CYST | TERATOMA |
|---|---|---|---|---|---|---|
| Not given | Died from pressure on pulm. artery | B | Cryptorchidism | Jöel considered tumor bronchogenic in origin; diagnosed it a teratoma | x | |
| Not clear | Died from pressure of tumor | B | | | | x |
| Not given | Apparently died from tumor | B | | Excellent microscopic and gross description with drawings of both | x | |
| | Death due to compression pulm. artery | B | | Information from Meltzer's paper | x† | |
| | Death prob. due to pression pulm. artery | B | | From Somolinos' paper, probably same case as Mathias | x† | |
| Not given | Death due to tumor | B | No | Pathologic description sketchy, no photomicrographs; probably bronchial in origin | x (?) | |
| Not available | Death prob. due to tumor | B | | | | x |
| Not given | Death due to tumor | B | Patent foramen ovale; patent ductus arteriosus | Authors did not speculate on origin; diagnosed teratoma; appears identical with ours | x | |
| Not given | Infection of tumor; septicemia | B | No | | | x |
| Not given | Died from tumor | B | | Author suggested tumor was bronchogenic in origin | x | |
| Not clear | Survived surgery; living and well | B | No | | | x |
| Root of aorta | Died from hemorrhage in attempted removal | B | Stunted growth | Could readily be bronchogenic in origin from description given | x | |
| Not given | Died from tumor | B | No | Called bronchogenic cyst by author | x | |
| Root of aorta | Death due to tumor | B | No | Compatible with bronchogenic origin (gastrointestinal potential) | x | |
| Root of aorta | Death due to tumor | B | Patent ductus arteriosus Twins | Compatible with bronchogenic origin (gastrointestinal potential) | x | |
| Not clear | Death due to tumor | M | No | | | x |

TABLE I

| AUTHOR-DATE AGE-SEX-HOW STUDIED | CLINICAL FINDINGS | X-RAY | SPECIAL STUDIES | SURGERY | PATHOLOGY, GROSS AND MICROSCOPIC | LOCATION-SIZE |
|---|---|---|---|---|---|---|
| Solomon,[32] 1951, 2 yr., F, autopsy | Superior vena cava syndrome | Large mediastinal shadow | No | Biopsy only | Resp. and squamous epith. with malignant degen. | Loc. same as ours, 2,100 Gm. |
| Fontaine,[3] 1954, 15 yr., F, operation | Found on routine chest x-ray | Same as ours | Tomography, angio. | Yes | Same as ours | Loc. same as ours, 7.5 × 4.5 × 2 cm. |
| Basora-Defilló,[12] 1956, 3 mo., F, autopsy | Dyspnea, cough cyanosis | Cardiac shadow filled chest | No | No | Same as ours | Loc. same as ours, 7 × 4 × 3 cm. |
| Miscall,[5] 1955, 3½ yr., F, operation | ? | Cardiac shadow filled chest | Angio. | Yes | Diagnosis, bronchogenic cyst | Loc. same as ours, 3 times the size of heart |
| Berg, 1957 42 yr., F, operation | See text | See text | See text | Yes | See text | See text |
| Dabbs, Peirce, Rawson,[4] 1957, 20 yr., F, operation | See text | See text | See text | Yes | See text | See text |
| Manion,[6] 1957, 44 yr., M, autopsy | Not known | Not known | Not known | No | Identical with ours | Not known |
| Mattingly,[33] 1957, 40 yr., M, clinicalDX. | Myocard. infarct. with recovery | Similar to ours | Angio., similar to ours | No | Not known | |

of Pathology. Both tumors were identical and fall in the category of bronchogenic cysts.[6] The relationship of these to cystic teratomas is confusing and they will be discussed together.

Jöel[7] first described this peculiar tumor in 1890 from an autopsy in a 14-year-old boy. Although he classified it as a "termatoma," his very exact description is identical with the 2 cases reported herein. Moreover, Jöel himself commented that the tumor was probably of bronchial origin. Maier,[1] in his excellent discourse on bronchogenic cysts of the mediastinum, alludes to Jöel's case of "bronchiogenic cyst," and we assume that he came to this same conclusion after translating Jöel's article.

We have been able to find, in reviewing the literature and from personal communications, 22 additional cases of cystic tumors within the pericardium (Table I). We are excluding the serous cysts (pleuropericardial) and the epithelial cyst or "simple cyst" as reported by Stockenius,[8] Kolatschow[9] and others.[10] These latter are uniloeular cysts lined by a single layer of epithelium. The epithelial lined simple cyst *may* be of bronchial origin but this is difficult to prove. The 2 cases herein reported are quite different from these but are practically identical grossly and microscopically except for their size. They were

CONT'D

| BLOOD SUPPLY | RESULTS OF AUTOPSY OR SURGERY | BENIGN OR MALIGNANT | ASSOCIATED CONGENITAL DEFECTS | COMMENTS | BRONCHOGENIC CYST | TERATOMA |
|---|---|---|---|---|---|---|
| Not clear | Death due to massive metastases | M | No | Compatible with malignant degeneration, bronchogenic cyst | x | |
| Not given | Patient survived and is well | B | Idiopathic epilepsy | This case identical with ours histologically and in position except smaller and more craniad | x | |
| Ascending aorta | Death due to tumor | B | No | Authors believed surgical removal was feasible; diagnosed teratoma, but appears identical with ours | x | |
| Root of aorta | Patient survived surgery and remained well | B | | | x | |
| Root of aorta | Survived, living and well | B | Congenital cataracts | See text | x | |
| Root of aorta | Survived, living and well | B | No | See text | x | |
| Not known | Not known | B | No | | x | |
| | | Presume B | No | ECG right posterior infarct suggests due to tumor; patient living and asymptomatic | x | |
| | | | | Total | 19 | 5 |

both located between the root of the aorta and the superior vena cava, deriving their blood supply from the former. They contained a number of different structures, all of which, in our 2 cases, are of bronchial origin (respiratory epithelium, stroma, lymphoid tissue, cartilage, squamous epithelium, vessels, and nerves). Focal necrosis and calcification is quite striking.

A careful review of the literature with reference to exact histologic descriptions and photomicrographs leads us to believe that many of the previous cases reported as "teratoma" are, indeed, identical with the cases reported herein. Jöel's case, for example, is clearly such a bronchogenic cyst, although called a teratoma by Jöel as he says "in accordance with Virchow's classification."[7] Jöel, himself, went on to write, "the whole structure of the tumor leads us to consider that its origin is from the bronchial tree." Those reported by Jellen and Fisher[11] and by Basora-Defilló and Lichtenberg[12] were also clearly bronchogenic tumors, although called "teratoma." A few of the reported cases of cystic tumors in this location, while resembling the bronchogenic tumors grossly and histologically in general, have structures such as hair and teeth and must be considered true teratomas,[13, 14, 15] although their gross appearance, location, and primary blood supply is strangely similar to the

bronchogenic cysts. In addition, there are several described which correspond closely to the clear-cut bronchogenic cysts but contain tissue described as "gastro-intestinal mucosa, pancreatic tissue, salivary gland tissue" or, in some instances, "groups of acini like salivary gland . . . some arrangements resembled pancreatic tissue."[16, 17, 18] Structures resembling ependyma and choroid plexus are also frequently recorded. In those instances where the respiratory structures predominante and where there are no structures clearly derived from ectoderm, we feel that the tumor is bronchogenic in origin.

Review of the available cases reveals no striking sex predilection. The ages range from newborn to 51 years, the majority being found in young people. Most of the cases are from autopsy specimens, the nature of the tumor being unsuspected before death. Usually the tumor was the cause of death by encroachment on vital structures. Of the 2 cases of malignant tumor in this group, one appears to be a malignant degeneration of a teratoma and the second probably of a bronchogenic cyst. Congenital anomalies were noted in 6 cases. These include cryptorchidism; patent foramen ovale and patent ductus arteriosus; stunted growth; idiopathic epilepsy; and congenital cataracts. In addition there are the 3 cases of extrapericardial bronchogenic cysts associated with a congenital defect of the pericardium. (See below.)

Beck's tumor, removed in 2 stages and those of Miscall and Fontaine appear to be the only tumors previously removed successfully. Fontaine's case and Miscall's case are the only previous ones studied by angiocardiography, and their findings were strikingly similar to ours. We are aware of another case, as yet unreported, of a young man in military service, who has had this diagnosis made clinically on the basis of angiocardiography, but this has not yet been confirmed by surgery.[33]

*Embryology.*[19-22]—In the very early embryo, the cardiac primordia and the pericardial coelom are paired lateral structures in contact with the ventrolateral aspect of the foregut, which is, at that time, continuous with the yolk sac. As the ventral aspect of the foregut closes, the cardiac primordia and the paired coelomic chambers fuse in the midline. Dorsally, however, the fusion is, for a time, incomplete, and there is a dorsal mesocardium. In the ten-somite embryo (17 to 19 days), the ventral foregut, therefore, is in continuity with the fused cardiac tube.

The "lung bud" takes origin from the floor of the pharynx at about 28 days and is rapidly converted into an elongated structure parallel to the foregut. It soon comes to lie between the foregut and the heart which are still connected by the dorsal mesocardium. Bronchial buds are present almost from the time the lung buds form and, until the obliteration of the dorsal mesocardium, these structures are in close proximity (Fig. 9). This region lies just medial to the right common cardinal vein, which later becomes cephalad and ventral. A nest of displaced cells from the rapidly developing bronchial buds

could easily locate in juxtaposition to the aortic portion of the truncus as the pericardial ridge over the common cardinal vein closes the pleuropericardial opening.*

Closure of the pleuropericardial folds at about the sixth week would generally trap any displaced cells within the pericardial cavity and this is the usual clinical finding. One might also expect to find variations in between represented by bronchogenic tumors in various locations and this is true.

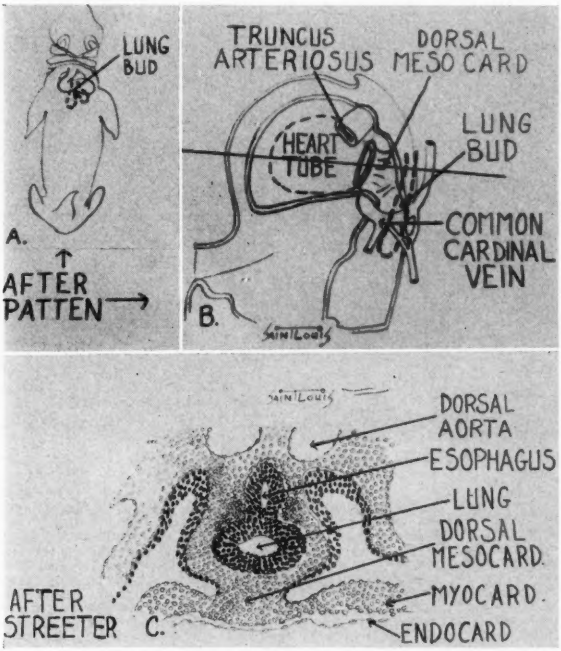


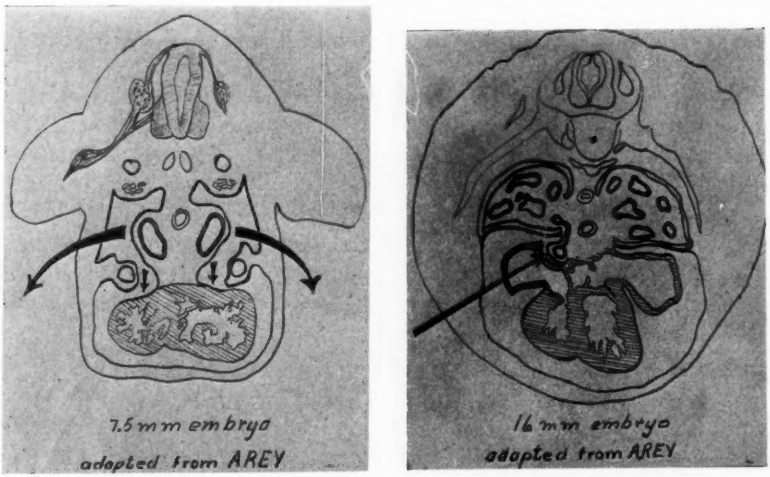

Fig. 9.—*A* and *B* (after Pattern,[19] Fig. 301), for orientation to show relation of "lung buds" to dorsal mesocardium. C (after Streeter,[21] Fig. 4, XV) shows quite clearly the proximity of the cells of the "lung bud" to the primitive heart through the dorsal mesocardium at this stage (26 to 28 days—3 to 6 mm.). Only a few cells separate the bronchial anlage from the dorsal mesocardium.

Chiari,[23] for example, has reported diverticula of the trachea; many authors have reported these tumors within the lungs, mediastinum,[1] presternal region,[2] and intrapericardially. The "missing links" which lend further credence to this theory are 3 cases[1, 6, 24] in which a typical bronchogenic cyst was found in juxtaposition to a congenital defect of the pericardium and a fourth case of a cystic teratoma "within the leaves of the pericardium."[35]

The origin of the lung bud from the pharyngeal part of the foregut suggests that inclusion of alimentary structures such as gastric mucosa and pancreas in some of these specimens does not preclude their bronchial derivation.

*The aortic root was definitely the point of attachment and source of major blood supply in 8 cases studied. In some, there has actually been a pedicle to the aorta.

Maier[1] expressed this same idea as follows: "An analysis of the literature reveals a difference of opinion concerning the stage in embryonic development at which aberrations occur which result in bronchiogenic mediastinal cysts. Although the wall of a typical bronchiogenic cyst is composed of structures similar to that in the bronchial wall, in an occasional case the components of the digestive tract are also found in the cyst wall."[1]




*A.* *B.*

Fig. 10.—(After Arey.[20]) This drawing illustrates a slightly different concept of origin of intrapericardial bronchogenic tumors. The growth of the lung, being primarily restricted to ventral and cephalad directions (*A*-large arrows) due to limitation by the body wall and septum transversum respectively, could direct itself medial to the common cardinal vein (and pleuropericardial fold), rather than lateral to this structure (*A*-small arrows). Closure of the pericardial coelom by approximation of the pericardial ridge to the mesocardium would irretrievably trap the anlage within the pericardial sac. The fact that these lesions occur predominantly on the right side could be explained by the more secure closure of the pleuropericardial opening on the right side by the persistence of the right common cardial vein as the superior vena cava. Conversely, the atrophy of the common cardinal vein could be correlated with persistent pleuropericardinal patency (pericardial defects), occurring predominantly on the left side.

## SUMMARY AND CONCLUSIONS

Bronchogenic cysts or "teratomas," as many of these have been diagnosed, occurring within the pericardium are unusual. To date we have found, from the literature and personal communication, 20 cases of these cystic tumors, to which we add the 2 cases reported herein. Both the tumors were quite large and both were removed successfully. The correct diagnosis was made preoperatively in one after clinical study including angiocardiography.

Although a few of those reported in the literature are clearly teratomas, most of them resemble closely the tumors which we are reporting and probably should be classified bronchogenic cysts. The predominating elements are cystic spaces lined with respiratory epithelium containing mucous glands, cartilaginous plates, and lymphoid follicles. Squamous epithelium and tissue derived from gastrointestinal primordia may also be present.

A plausible embryologic explanation for the occurrence of bronchogenic cysts within the pericardium is advanced. During the 28- to 34-day period the "lung buds" are in close contact with the dorsal mesocardium and heart tube. It appears logical that cells having respiratory (and gastrointestinal) potential may become displaced, develop in juxtaposition to the aorta and superior vena cava, and are sealed within the pericardium by fusion of the pleuropericardial fold. The few cases which are strikingly similar grossly but which contain tissue clearly of ectodermal origin must be diagnosed teratomas. We have no adequate explanation for their similarity nor their occurrence here unless it be by the same mechanism, with metaplasia of the primordial cells.

The x-ray configuration and the deformity produced on angiocardiography appears characteristic. Surgical attack without proper knowledge of the intimate association of these tumors with the cardiac chambers and the great vessels may be disastrous. Exact diagnosis and precise localization in relation to the anatomy of the heart preoperatively should lead to successful removal in most instances.

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## DISCUSSION

DR. LAURENCE MISCALL, New York, N. Y.—The case I wish to present was mentioned by the previous speaker. This 2½-year-old female child was treated at the age of one year as a congenital cardiac, because of a grossly enlarged cardiac silhouette. After 16 months, when she began to have severe respiratory infection and dyspnea, she came to The New York Hospital.

(slide) This admission plate demonstrates well the great increase in the cardiac outline.

(slide) On this angiocardiogram one can see the marked displacement of the superior vena cava and its relation to the right border of the heart.

(slide) Here can be seen similar displacement of the right atrium, and the right ventricle with the pulmonary artery originating almost at the left thoracic wall.

(slide) The pressure on the left atrium is visible in the center.

(slide) Here it can be seen nicely that this tumor was so large that it caused displacement of the aorta to a position far to the left of normal.

(slide) This tumor was so large that with the chest open through a sternal splitting incision, one could not see the heart. I think this accounted for the absence of all cardiac sounds anteriorly. Posteriorly, a short systolic murmur was heard by some observers.

(slide) This is the tumor that was removed, together with a piece of the right atrium. No other blood supply was found.

(slide) Here are two sections, this one rather typical of what might be found in a tumor of bronchogenic origin.

(slide) But this other section is rather suggestive of gastric origin.

(slide) This is the child's postoperative plate. The inference that such angiocardiographic findings may be typical of these is not true. I would like to show two quite similar x-rays, however, on a different type of lesion.

(slide) Here is the admission plate on another child who entered with a somewhat similar story.

(slide) Here one notes quite comparable displacement of the aorta and the pulmonary artery.

(slide) Here the right atrium is flattened and the curvature of the superior vena cava altered. This patient did not have an intrapericardial bronchogenic cyst, but a teratoma, extrapericardial in position.

# NEUROMUSCULAR IMBALANCE OF THE ESOPHAGUS ASSOCIATED WITH HIATAL HERNIA AS STUDIED BY MEANS OF CINEFLUOROGRAPHY AND INTRALUMINAL PRESSURE RECORDINGS

FREDERICK S. CROSS, M.D., EARLE B. KAY, M.D., AND GEORGE F. JOHNSON, M.D.
(BY INVITATION)
CLEVELAND, OHIO

THE present report is part of an extended and continuing project originally designed to evaluate cinefluorography as a diagnostic, investigative, and teaching tool in the study of certain neuromuscular diseases of the esophagus. Utilizing this technique, esophageal studies can be recorded on motion picture films with intensifying systems, so that dynamic changes can be recorded in a dynamic manner, rather than as static x-ray film which frequently miss or inadequately record the important facts. It became apparent, however, that cinefluorography alone was insufficient, and that it had to be correlated with a careful history, routine fluoroscopic and x-ray studies, esophagoscopy when indicated, and intraesophageal pressure studies as well, in order to evaluate completely problems related to changes in esophageal motility. Interest was stimulated in studying neuromuscular changes in the esophagus associated with hiatal hernia because of the frequent observation of dysphagia far out of proportion to the size of the hernia or to the apparent changes in the esophagus, such as esophagitis and stricture.

## METHODS

A total of 55 patients with esophageal problems falling into the following categories have been examined: neuromuscular failures, segmentation, achalasia (cardiospasm), diverticula, and hiatal hernia. Neuromuscular failure and segmentation have been the subject of a previous report,[1] but will be discussed briefly here since the changes found in these conditions are basic to an understanding of those taking place in association with hiatal hernia.

Each patient was evaluated with routine fluoroscopy and x-ray studies, esophagoscopy when feasible, intraluminal pressure studies, and cinefluorography. Motion pictures were taken by means of a cinefluorographic unit comprised of a grid, fluoroscopic screen, right-angle mirror, bellows, F. 71 Wray lens, 35 mm. camera, magazine, and commutator. The commutator synchronizes the x-ray exposure, so that exposure is limited to the time that the film is in place. In these studies, maximum exposure was limited to 14 seconds. The patient was placed in the left posterior oblique position for examination of the thoracic

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esophagus and thin liquid barium paste was swallowed as a large single bolus. The movie was started when the order was given for the patient to swallow. Studies of the cervical esophagus were obtained in the lateral position, the patient swallowing a thick barium paste one second after the movie camera was started.

The esophageal motility studies were carried out with the patient in the supine position on the fluoroscopic table. Polyethylene catheters 1.77 mm. I.D., with 5 side holes in the distal portion were used to record the esophageal pressure. Radiopaque markers were placed at the tips of the catheters so that tube end localization for the accurate placement of the pressure recording tube in the various levels of the esophagus could be ascertained fluoroscopically and by x-ray when desired. The catheters were filled with water and connected to a model T23D Statham pressure transducer, in turn connected to a cathode ray recorder. Two single lumen tubes were used when simultaneous pressure recordings were desired at different levels of the esophagus. The pressure transducer was localized at the same level as the catheter lying in the esophagus and the pressure recordings were standardized by means of a mercury manometer. Initiation of swallowing was marked by a signal at the onset of the elevation of the larynx. The majority of pressure recordings were made with dry swallows.

## BASIC MOTILITY PATTERNS

*Normals.*—Normal patterns of esophageal activity have been established by cinefluorography and intraluminal pressure studies which correspond to those described by other investigators.[2, 3, 4] Normal pressure patterns obtained in the pharynx, cricopharyngeal area, the upper one third, middle one third and lower one third of the thoracic esophagus, the vestibular portion of the esophagus, and the stomach are illustrated in Fig. 1. Food is ejected into the upper esophagus by the glossopharyngeal pump mechanism through the cricopharyngeus muscle which is tonically closed in the normal esophagus, and must relax at the onset of swallowing to allow passage of the bolus. The high base pressure produced by the tonic closure of the glossopharyngeal muscle is shown. The negative phase of the complex is produced when the cricopharyngeus muscle relaxes reciprocal to the increase in pharyngeal pressures as swallowing is initiated. This relaxation phase is followed by the contraction phase as the peristaltic wave passes through the muscle area. It has been an interesting observation that the cricopharyngeal pressure patterns have, in general, reflected the degree of activity present in the rest of the esophagus. There is a progressive increase in the peristaltic interval as the recording catheter is passed down the esophagus. This ranges from 0 seconds in the pharynx to 4.5 to 5.0 seconds in the lower esophagus. Base pressures also vary in the different segments, ranging from a positive pressure in the pharynx and cricopharyngeal area, to a negative pressure in the thoracic esophagus, to a fluctuating positive-negative pressure in the vestibular area, where the positive intra-abdominal pressure begins to have an effect, to a positive pressure in the stomach. Respiratory variations in the pressure curves in the thoracic esophagus and in the vestibular area are

noted. The sites of the sphincteric action supposedly possessed by the constrictor cardae muscle and the inferior esophageal constrictor have not been localized by elevated base pressures as have been recorded for the cricopharyngeus muscle. The amplitude of recorded peristaltic contractions averages about 30 mm. Hg in the thoracic esophagus, there being some variation at the different levels. Recorded peristaltic waves in the vestibular area are low and frequently may be absent. Generally, they are higher in the cricopharyngeal and pharyngeal areas than in the thoracic esophagus.

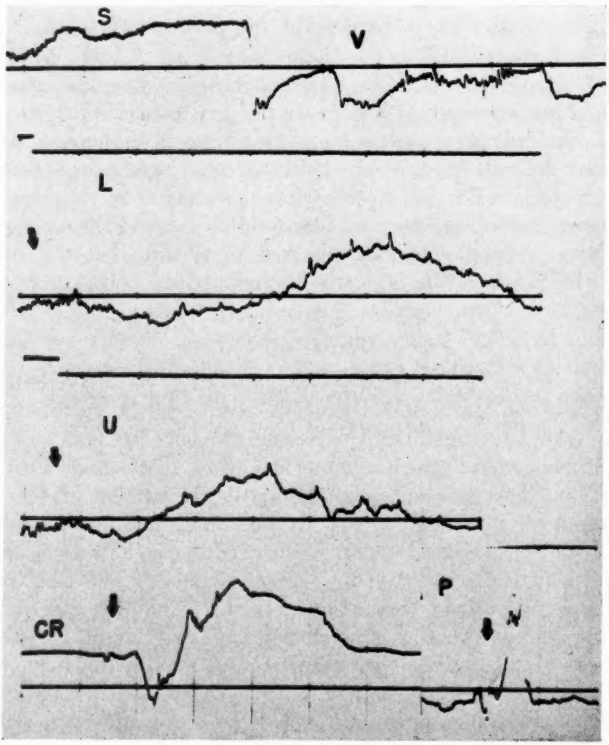


Fig. 1.—Normal intraesophageal pressure tracings. The arrow indicates the time of the swallowing. The letters indicate the level of the tip of the recording catheter in the esophagus; *S* = stomach, *V* = vestibule, *L* = lower one third, *M* = middle one third, *U* = upper one third, *CR* = cricopharyngeal area, *P* = pharynx. The intervals are one second.

*Neuromuscular Failure.*—Two basic patterns of neuromuscular imbalance have evolved which may be primary, with no apparent underlying etiology, or may be secondary to the presence of some other condition, such as a hiatal hernia. The first is basically one of hypotonicity and hypoactivity and has been termed neuromuscular failure. As a primary entity, neuromuscular failure occurs more frequently in older debilitated people. Typical intraesophageal pressure tracings from such a patient are illustrated in Fig. 2. Low base pressures are

recorded in the cricopharyngeal area indicating poor muscle tone, and peristaltic contractions in this region are poor. The activity in the cricopharyngeal area reflects the changes in the remaining esophagus, for there is virtually a complete absence of peristalsis found at all levels of the thoracic esophagus.

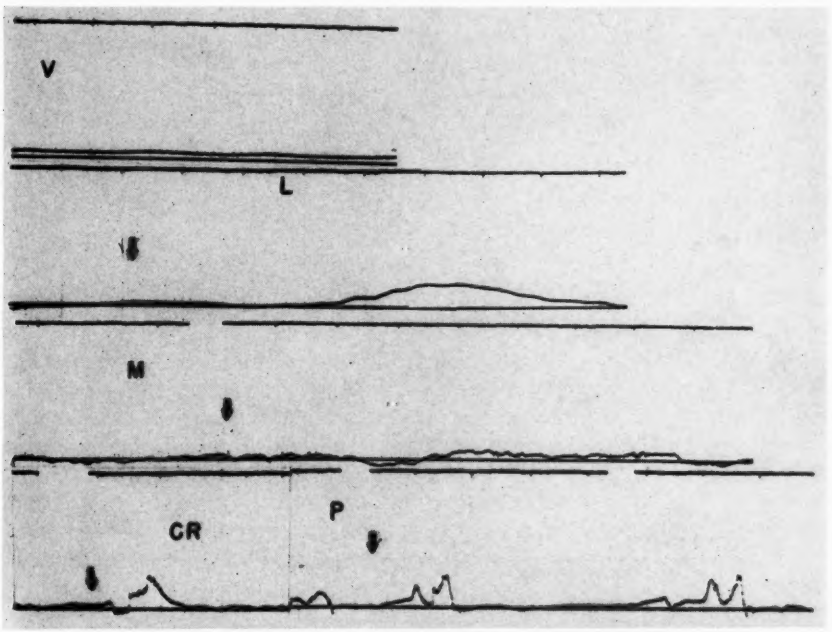


Fig. 2.—Neuromuscular failure. The symbols are the same as in Fig. 1. There is complete lack of peristaltic activity at all levels of the thoracic esophagus. There is a decreased base pressure and diminished contraction wave in the cricopharyngeal area as well.

This hypoactivity of the esophagus in neuromuscular failure is reflected in the fluoroscopic and cinefluorographic studies of the esophagus, which reveal a great delay in the passage of barium through the esophagus to the stomach. This is especially true with the patient in the supine position, since clearing of the esophagus with the patient lying down is unaided by the gravity effect. Transit time through the esophagus may be more than 3 minutes. Frequently mis-swallowing of barium is seen, and regurgitation of esophageal contents through the hypoactive cricopharyngeus muscle with subsequent aspiration occurs.

*Segmentation.*—The second pattern of esophageal neuromuscular imbalance has been termed segmentation and represents a hyperactive, hypertonic condition of the esophagus. Intraesophageal pressure studies, as illustrated in Fig. 3, may show base pressures in all segments to be high. This is particularly true in the cricopharyngeal complex reflecting the increased tone of the muscle. The

negative phase due to muscle relaxation in front of the advancing peristaltic wave is less than normal, and the positive phase of the cricopharyngeal complex is exaggerated and prolonged. The peristaltic waves recorded in the upper one third to one half of the esophagus may be normal, but are more often diminished or absent. On the contrary, the peristaltic waves in the distal two

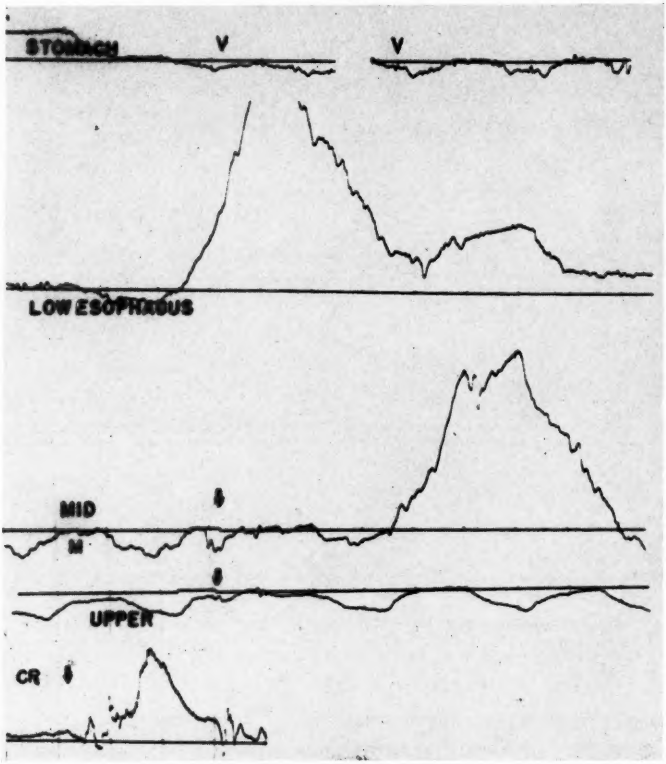


Fig. 3.—Segmentation. The symbols are the same as in Fig. 1. Peristaltic activity in the lower two thirds of the esophagus is exaggerated. There is a double hump effect in the relaxation phase of the lower third curve. Esophageal contraction in the upper third of the thoracic esophagus is diminished. The cricopharyngeal complex shows a diminished relaxation phase but, in this instance, the contraction phase is fairly normal.

thirds of the thoracic esophagus are high and prolonged with an average peristaltic pressure three times that recorded in the normal individual. A double or triple hump effect may be demonstrated during the relaxation phase of the curve due to the incoordinated segmental contractions. The vestibular area of the esophagus may likewise show elevated peristaltic waves, and they are present more frequently than in the normal individual. In effect, the cricopharyngeal area and the lower two thirds of the esophagus are hyperactive, while the upper one third to one half of the esophagus is normal or hypoactive.

This altered esophageal physiology is reflected in the changes found in the barium swallow recorded by routine fluoroscopy or cinefluorography. Although there is normal progression of barium into the proximal one third of the esophagus, there is delay of further progress at about the junction of the upper and middle thirds of the esophagus. With barium in the lower half of the esophagus, segmentation appears as a generalized tone increase; the segmental areas of narrowing occurring at the height of the contractions. Incoordinated churning of the barium in the lower esophagus is seen with considerable retrograde expulsion of barium into the proximal one third of the organ. This hyperactivity in the lower thoracic esophagus with normal or hypoactivity in the upper results in a functional obstruction giving rise to significant symptoms of dysphagia. Other common complaints are tightness in the throat and difficulty in initiating swallowing, both probably a reflection of the hyperactive cricopharyngeus muscle.

### HIATAL HERNIAS

Hiatal hernias were originally included in this study not only in an attempt to determine the basis of dysphagia associated with hiatal hernias, but also to see if a small hiatal hernia, difficult to diagnose by conventional methods, might be demonstrated more adequately by cinefluorography. It soon became apparent that the problem of hiatal hernia is not limited to the lower esophageal segment, but that neuromuscular imbalance of the entire organ is an important factor in the over-all picture. Most of the studies were carried out on patients with concentric or direct types of hiatal hernia, since the incidence of esophageal complications seems to be higher in this type.

It is not possible to discuss hiatal hernias as a uniform group, since important secondary manifestations of the hernia may bear no relation to its size. In other words, marked regurgitation and esophagitis may be associated with a barely perceptible hernia, while a larger one is essentially asymptomatic. In all hernias the cardiac sphincter mechanism is altered to some extent allowing regurgitation of acid-peptic juice into the lower esophagus. In uncomplicated, mildly symptomatic hernias, this alteration in the cardiac sphincter mechanism may be of no consequence, and regurgitation of acid-peptic juice from the stomach minimal or absent. In complicated, symptomatic hernias, a breakdown of the cardiac sphincter mechanism exists, and regurgitation of acid-peptic juice into the lower esophagus becomes of major importance. A greater burden is then placed on the inferior esophageal sphincter as the only remaining protective mechanism against free regurgitation of acid-peptic juice into the upper esophagus. Following the repeated insult of acid-peptic juice extensive esophagitis occurs and a lower esophageal stricture may develop. With stricture formation, the competency of the inferior esophageal constrictor is overcome and there is unimpeded regurgitation into the upper levels of the esophagus. Although hiatal hernias can be divided roughly into the three stages discussed above, the term does not imply that one stage necessarily progresses to another; however, it has been possible to trace the development of a small uncomplicated hernia into one with the severe complication of esophageal stricture.

Intraluminal pressure studies have been particularly helpful in the study of hiatal hernias, both as a diagnostic aid and as a means of studying associated altered esophageal physiology. Although normal esophageal motility may exist, especially in those individuals with small uncomplicated hernias, more frequently the abnormal motility pattern previously described under neuromuscular failure

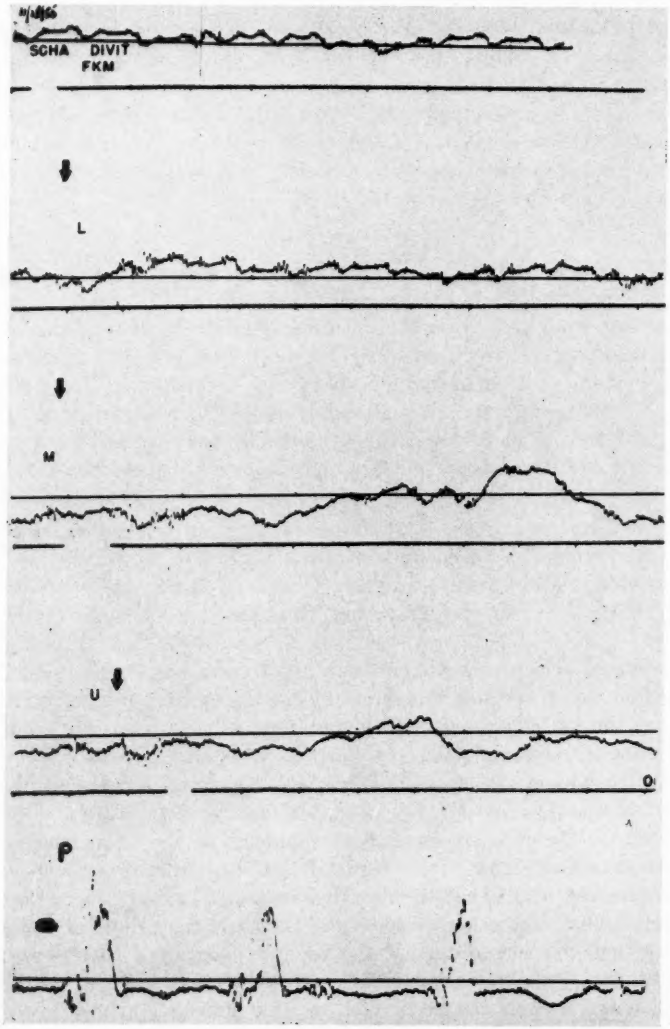


Fig. 4.—Esophageal pressure studies from the patient whose roentgenograms are shown in Fig. 6. Note the positive resting pressure recording in the lower esophageal area. There is a decrease of esophageal activity at all levels of the thoracic esophagus. The pharyngeal complex is normal.

is found in association with hiatal hernias. Figs. 4 and 5 illustrate such a study in the patients whose roentgenograms are seen in Figs. 6 and 7. There is diminution or absence of peristaltic activity throughout the thoracic esophagus. The cricopharyngeal complex is essentially normal, which differs from the hypoactive cricopharyngeal complex shown in Fig. 1. The intraesophageal pressure studies in patients with hiatal hernia show a second significant change which has been of further aid in the evaluation of hiatal hernias. The transitional zone between the positive base pressure, normally occurring in the stomach, and the negative base pressure in the esophagus is found at a higher esophageal level than usual.

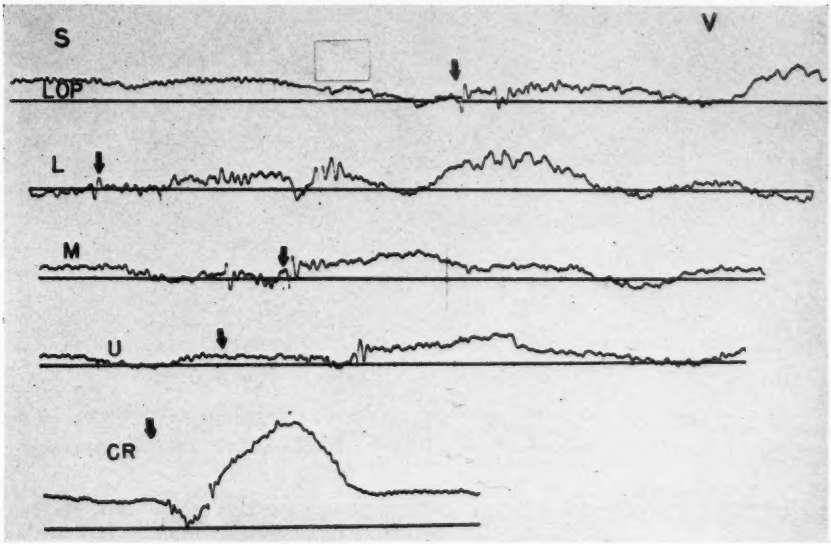


Fig. 5.—Pressure studies from the patient whose roentgenograms are shown in Fig. 7. The transitional zone between stomach and lower esophagus is gradual. The cricopharyngeal complex is somewhat exaggerated.

This indicates that the positive intra-abdominal pressure is transmitted above the diaphragm, probably into the herniated portion of the stomach, or up the esophagus past an inferior constrictor made incompetent by stricture formation. By correlating the position of the catheter tip fluoroscopically with the tracing obtained on the pressure recorder, the presence of a small hiatal hernia may be ascertained. The sharp drop in base pressure as the catheter is withdrawn from the stomach into the vestibular portion of the esophagus in a normal individual is well illustrated in Fig. 1. In contrast, the pressure tracings from individuals with hiatal hernia show a positive base pressure at a level that would normally be lower esophagus (see Figs. 4 and 5). The transmission of the intra-abdominal pressure up the esophagus is even more marked in patients with a stricture of the lower esophagus in which the action of the inferior esophageal constrictor is lost (Fig. 8). As shown in Fig. 9, the positive base

Fig. 6.

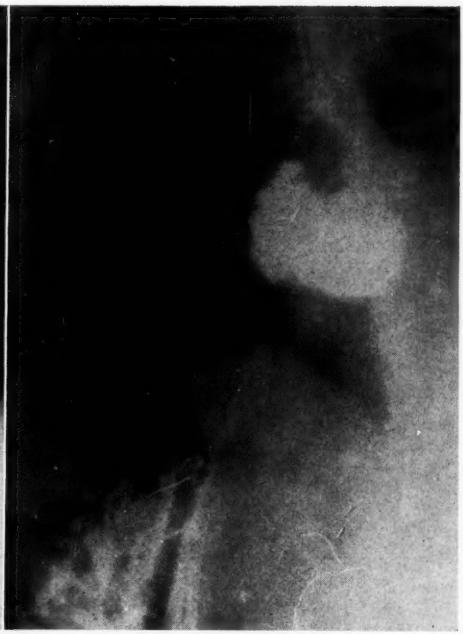

Fig. 7.

Fig. 6.—A small hiatal hernia associated with a lower esophageal diverticulum. The pressure studies in Fig. 4 are from this patient.

Fig. 7.—Hiatal hernia roentgenograms showing the hiatal hernia in a patient whose pressure tracings are shown in Fig. 5.

Fig. 8.—Roentgenogram from a patient with lower esophageal stricture associated with a long-standing hiatal hernia and duodenal ulcer.

pressure is recorded in the upper levels of the esophagus past the strictured area. Note that there is complete absence of esophageal activity as in neuromuscular failure; and again, the cricopharyngeus muscle exhibits a fairly normal response. In no instance has the hyperactive motility pattern associated with segmentation been found in patients with hiatal hernia.

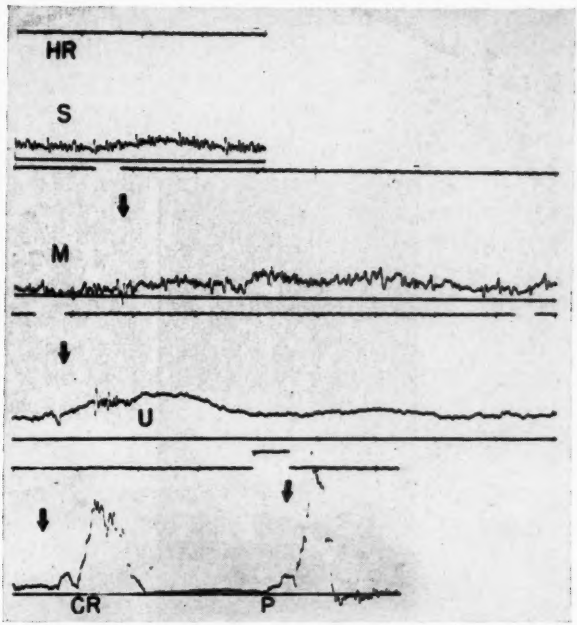


Fig. 9.—Intraluminal pressure studies from a patient with long-standing lower esophageal stricture secondary to hiatal hernia. Note the complete absence of peristaltic contractions at all levels except in the cricopharyngeal area. The positive base pressure is transmitted to the upper levels of the esophagus.

Cinefluorography has been of some value in outlining small, moderately symptomatic hiatal hernias in which the differential diagnosis between a hiatal hernia, a prominent vestibule, or a phrenic ampulla, is difficult or impossible by conventional means. Localization of the esophagogastric junction in relation to the diaphragmatic hiatus may be made at times by esophagoscopy. Cinefluorography is of value in that the barium swallow is recorded as a movie, which may be studied separately to identify the anatomic landmarks in the area of the terminal esophagus important in outlining a hiatal hernia.

With the repeated regurgitation of acid-peptic juice into the lower esophagus the inferior esophageal constrictor may become hyperactive, resulting in a so-called "contraction band" which may be seen on routine roentgenograms (Fig. 10), but is more readily demonstrated by cinefluorography. The presence of the hyperactive inferior esophageal sphincter is an important clue to the presence of a small hiatal hernia. The hyperactivity of the inferior esophageal

constrictor seen on cinefluorography may be associated with normal peristaltic activity in the remainder of the esophagus, or with the hypoactivity seen with neuromuscular failure. In either event, esophageal transit time is slowed. Regurgitation of stomach contents into the lower esophagus has been well demonstrated by cinefluorography, especially in one patient with a stricture of the lower esophagus. The esophagus above the strictured area seems to empty more or less passively through the strictured area much as sand through an hourglass. The most consistent changes in esophageal activity associated with hiatal hernia recorded by cinefluorography have been hyperactivity of the inferior esophageal constrictor and hypoactivity of the upper esophagus as seen in neuromuscular failure.

Fig. 10.—Roentgenogram from a patient with a "contraction band" in the lower esophagus due to a hyperactive inferior esophageal constrictor associated with a small hiatal hernia.

## DISCUSSION

Although the concepts of neuromuscular imbalance of the esophagus discussed here are not entirely new, they are still not generally appreciated except perhaps in relation to changes associated with achalasia of the esophagus. What we have called segmentation has been described previously as diffuse spasm of the lower esophagus by Moersch and Camp,[5] Schmidt,[6] and Creamer, Donoghue, and Code.[7] Kramer and Ingelfinger[8] have described neuromuscular failure in relation to scleroderma, and Lorber and Shay[9] have reported a dysrhythmia of esophageal motor activity noted fluoroscopically in association with a hiatal hernia.

With the basic concepts of altered esophageal motility established, cinefluorography and intraluminal pressure studies have been valuable adjuncts in the evaluation of patients with hiatal hernias. From these studies it has become apparent that the entire esophagus may be altered in association with the hiatal hernia. The intraesophageal pressure studies have shown significant changes in peristaltic activity and intraesophageal pressure relationships. This altered peristaltic activity undoubtedly accounts for the dysphagia frequently associated with hiatal hernia which is out of proportion to other objective findings. The alteration in intraesophageal pressures with the positive intra-abdominal pressures being transmitted into the lower esophagus points out the basis for the vicious cycle of acid-peptic regurgitation into the esophagus.

Cinefluorography records, as a permanent dynamic record, changes in esophageal motility frequently seen by the fluoroscopist, but heretofore unrecorded for the clinician. Transit time through the esophagus is delayed either in the lower esophagus due to hyperactivity of the inferior esophageal constrictor, or in the upper esophagus due to a generalized hypoactivity in esophageal motility. If these changes are noted by fluoroscopy or cinefluorography the presence of an undetected hiatal hernia should be suspected. In addition, cinefluorography has been a valuable teaching tool in demonstrating the functional anatomy of the esophagus at all levels, both in health and disease.

## SUMMARY

1. The use of cinefluorography and intraluminal pressure tracings for evaluating neuromuscular changes in the esophagus in association with hiatal hernias is discussed.

2. Normal motility patterns, as well as those found in neuromuscular failure and segmentation, are described.

3. The basic pattern obtained in patients with hiatal hernia has been hypoactivity of the esophagus, elevated base pressures, especially in the lower end, and hyperactivity of the inferior esophageal sphincter.

4. It is felt that cinefluorography and intraluminal pressure studies are valuable as investigative, diagnostic, and teaching aids in neuromuscular diseases of the esophagus.

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## Discussion

DR. CAMERON HAIGHT, Ann Arbor, Mich.—What is the relative amount of specific radiation received?

DR. FREDERICK S. CROSS, Cleveland, Ohio (Closing).—The amount of radiation received in this type of an examination obviously varies with the size of the individual and the size of the field. Our radiologists have figured out exposure in cineangiography, and certainly radiation exposure in cinefluorography of the esophagus should be comparable. At an output of 336 r per minute (skin) for a maximum period of 15 seconds, the exposure is equal to 84 r in the average adult. For comparison with these figures, cardiac catheterizations are allowed from 10 to 15 minutes of fluoroscopic time at 15 to 20 r per minute. These same exposure factors obtain for diagnostic fluoroscopy. From the above information it can be concluded that the radiation hazard to the patient from primary radiation seems acceptable, and actually it is not as great as that hazard which is unquestionably accepted for many other procedures. There are individuals who are doing this type of work with the image amplifier which gives a great deal less radiation exposure but, since it has a smaller field, it is necessary to limit the examination to specific segments of the esophagus rather than to the entire esophagus at once. We also can cone down on specific areas of the esophagus and thereby eliminate a certain amount of radiation exposure. Because of the question of radiation exposure, we have not repeated any examinations to date, but plan to do so in the near future, especially in older patients.

# ESOPHAGEAL HIATUS HERNIA OF THE DIAPHRAGM

## An Analysis of Surgical Results

George H. Humphreys II, M.D., Jose M. Ferrer, Jr., M.D. (by invitation), and Philip D. Wiedel, M.D. (by invitation)
New York, N. Y.

### INTRODUCTION

HERNIAS through the inguinal canal, one of the commonest afflictions of mankind, have been well understood since the classical anatomic studies of over a century ago. Elective radical repair as the treatment of choice has been accepted since the controversies of the turn of this century established, by adequate follow-up studies, that effective methods were available. Hernias through the esophageal hiatus, on the other hand, received little attention until recently and are still poorly understood by many physicians. The anatomy is inadequately described in many standard texts, and the symptom complex which results from the disturbed physiology of swallowing is frequently misinterpreted. As was the case with inguinal hernias 60 years ago, most physicians still hesitate to suggest operation for a hiatal hernia even when it has been recognized to exist, feeling that it is a common incidental finding of little significance, and attributing its symptoms to other causes, or finding none, to neurosis. Only when obviously incapacitating complications develop is repair resorted to, and even then with misgivings, because the risks of the procedure and the likelihood of recurrence are thought to be high.

Yet there is now no longer any need for these misunderstandings, misinterpretations, hesitations, or misgivings. Careful anatomic reviews have made clear the anatomy of the esophageal hiatus in the diaphragm, demonstrating its considerable individual variations, and indicating the part it plays in holding the gastric cardia in place and controlling reflux from the stomach into the esophagus. The more complex intrinsic mechanisms of closure of the gastric cardia have also been extensively studied. Although here the findings of anatomist, endoscopist, and roentgenologist are more difficult to correlate, there is general recognition that incompetence of the cardia follows if, sliding through the hiatus, it loses its acute angle of entrance. No one, after reading Allison's description,[2] should fail to recognize the symptoms which result. Furthermore, several methods of repair which are comparatively free of risk and at least immediately effectual have been described. These methods differ considerably in approach and technical detail, but the mistaken theories and techniques which gave poor results in the past have now been replaced by recognized principles of anatomy and physiology upon which all successful methods are based.

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In spite of the many papers on the subject in recent years, however, it is still difficult to determine the actual long-term results of modern methods of repair of hiatal hernia. Most writers have been concerned with anatomy, symptoms, technical methods, or immediate results. It has seemed worth while, therefore, to review the long-term results obtained in a group of patients operated upon by several surgeons in a single institution, and covering the transitional period during which a successful method emerged. This method is thought to be based upon sound principles, and has now been in use for 8 years. It differs from others in simplicity and seems to have stood the test of time. Our purpose, however, is not so much to describe a method, as to bring out some of the commoner pitfalls and misconceptions, as well as the possibilities of successful management of hiatal hernia, as demonstrated by end results 2 or more years after operation.

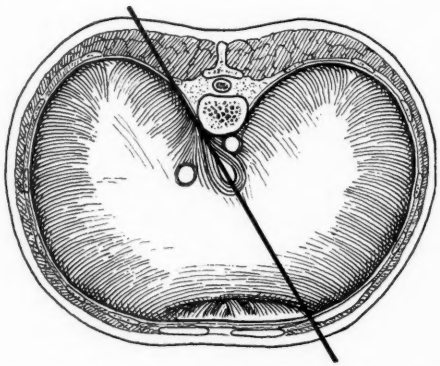

Fig. 1.—A diagram of the diaphragm viewed from above, showing the muscular sling of the esophageal hiatus formed from the right crus. The oblique anteroposterior axis is indicated by the heavy line, which is also the plane of the diagrams in Figs. 2 and 3, as well as the view seen by the surgeon in a left posterolateral thoracic approach, as shown in the drawings of the operative procedure.

## ANATOMY—NORMAL AND PATHOLOGIC

The anatomy of the esophageal hiatus has been carefully studied and well discussed by many authors.[1, 2, 3, 8, 9, 15, 18, 27] It has been clearly established that the esophageal hiatus of the diaphragm is formed by the right crus, which normally surrounds the esophagus just above the cardia to form an oval slinglike aperture. In rare instances, a few fibers from the left crus cross over and enter into the formation of the esophageal hiatus. Ordinarily, however, the left crus confines itself to joining the right crus in the formation of the aortic hiatus. The "pinchcock" action of the hiatus is dependent on its slinglike configuration and contributes to the prevention of reflux from stomach to esophagus (Fig. 1).

An important anatomic structure at the hiatus is the phrenoesophageal ligament.[2, 13] This is in reality an extension upward through the hiatus of the fibers of the infradiaphragmatic layer of the transversalis fascia of the abdomen. These fibers invest the cardia and lowermost esophagus in a sheath and are inserted into the adventitia of the esophagus above the cardia. The

phrenoesophageal ligament varies in strength and thickness from patient to patient, as indeed does the transversalis fascia encountered in inguinal hernia repair. When this ligament is well developed, it is a major factor in holding the cardia below the diaphragm.

A second anatomic "anchor," which is thought to play a part in holding the cardia in its normal infrahiatal position, is the left gastric artery and its mesentery.[3] This structure normally stretches very little, even under tension, and it effectively tethers the cardiac portion of the lesser gastric curve to the posterior abdominal wall.

Reflux through the cardia is normally controlled also by intrinsic mechanisms.[14, 16] The presence of a flap valve of mucosa and gastric wall has been postulated, and an operation to reconstruct or enhance this valve has been described.[17] Probably more important is the so-called cardiac sphincter muscle. This thickened band of the circular muscle coat of the esophagus is aided in its action of keeping the cardia closed by a sling of gastric musculature which surrounds the esophagogastric junction just below the cardia.[3] In addition, the terminal esophagus or epiphrenic ampulla helps to prevent reflux.[11] Whatever the intrinsic mechanisms may be, it is generally agreed that they are effective only when the normal acute esophagogastric angle at the cardia is preserved.[7, 23]

When a hernia occurs through the esophageal hiatus, a number of factors are present. The difference between intra-abdominal and intrathoracic pressure is one factor. Increased intra-abdominal pressure due to obesity, pregnancy, or the acute increase of a sudden blow appears to play a real part in increasing a herniation once it has begun, although it may be questioned as an original cause. Similarly the inherent tendency of the esophagus to shorten, and the sucking effect of the inspiratory phase of respiration are secondary factors. Congenital variations in the construction of the crural sling, or weakening of the fibers with age, seem more likely original factors in the common sliding hernia. A pre-formed peritoneal sac, sometimes called the infracardiac bursa, and considered to be a persistence of the pneumatoenteric recess of the embryo, may protrude upward through the hiatus and predispose to paraesophageal hernia.[26, 27] Finally the length of the esophagus is a factor. In the usual sliding hernia, the esophagus becomes shortened secondarily. In rare instances, the esophagus may be congenitally short. In this case the stomach has never fully descended below the diaphragm.[3, 20, 28] Rarely, there may be no crural fibers between the esophagus and aorta, a condition termed "esophago-aortal hiatus hernia."[1]

All of these anatomic factors contribute to the formation of a hiatal hernia. The etiological significance and importance of each factor vary in different cases. Successful repair depends upon knowledge of the varied anatomic types, the ability of the surgeon to recognize and deal with each, and an understanding of methods to prevent gastric reflux.

## CLASSIFICATION AND DEFINITION OF TYPES

There are 3 main types of acquired hiatal hernias. The first, and by far the commonest, is the sliding hernia. The second type is the paraesophageal.

The third type is the combined or composite hernia which is a combination of the first and second.

Sliding hernias occurred in our series in 87 cases. In this type, the cardia lies at a variable distance above the diaphragm and the cardiac end of the stomach slides up retroperitoneally and retropleurally. There is no true peritoneal sac present in these hernias, but there may be a fold of peritoneum which has been pulled up by the sliding stomach. As in all sliding hernias, this peritoneal fold is formed by the rolled back peritoneum of the sliding viscus (in this case the stomach), which is loosely attached where it is reflected onto the undersurface of the diaphragm. In very large sliding hernias, this "sac" may become large enough to allow gastrocolic omentum or even colon to pass up into the chest. In all, the hiatus is dilated, the phrenoesophageal ligament is stretched and thinned, and the left gastric blood vessels are long and lax. The esophagogastric angle has become obtuse, the mucosal valve no longer exists, and the cardiac sphincter alone cannot prevent gastric reflux with its resultant symptomatic esophagitis. The esophagus is short for one of two reasons. The shortening may be due either to muscular contraction of the longitudinal muscle coat of the esophagus or to scarred fibrous contraction due to advanced inflammatory changes in the esophagus wall. The former is fortunately much more frequent, since in these cases the esophagus readily stretches back to its normal length when the cardia is replaced below the diaphragm. In the latter, it is difficult to regain esophageal length and the hernia is less susceptible to satisfactory repair (Fig. 2).

Recently, a special subtype of the sliding hernia has been recognized and described. This is the esophago-aortal hernia[1] which protrudes through a common hiatus between the esophagus anteriorly and the aorta posteriorly. It is not entirely clear whether this is a congenital failure of formation of separate esophageal and aortic hiatuses or whether the muscle fibers between the two hiatuses are destroyed by the pressure of the enlarging hernia. The latter seems the more logical explanation. In this series, there were 2 of the esophago-aortal type of hiatal hernias.

A paraesophageal hiatal hernia was present in 6 cases. In this hernia, the phrenoesophageal ligament and the left gastric vessels are firm and holds the cardia and the lesser curvature of the stomach below the hiatus. The fundus and greater curvature of the stomach protrude up beside the cardia and esophagus into a peritoneal sac in the mediastinum. There may or may not be a small band of muscle or fibrous tissue between the cardia and the paraesophageal hernial sac.[27] If such a muscle band is present, the hernia is more correctly called parahiatal. More often, the hiatus is dilated, but the esophagus is of normal length.

The combined or composite type of hernia was seen in 3 cases in this series. Frequently mistaken for a true paraesophageal hernia, it differs from the latter in that the cardia lies above the diaphragm though the fundus and greater curvature have rotated upward to an even higher position, so that a sac containing other abdominal viscera may be present. It probably is the result

of a herniation which, beginning as an ordinary sliding hernia, has progressed by disproportionate weakening of the anterolateral muscle fibers of the hiatal sling, as compared to the posteromedial crural fibers, to permit the major herniation to take place anterior to the cardia. These cases are therefore grouped with the sliding hernias.

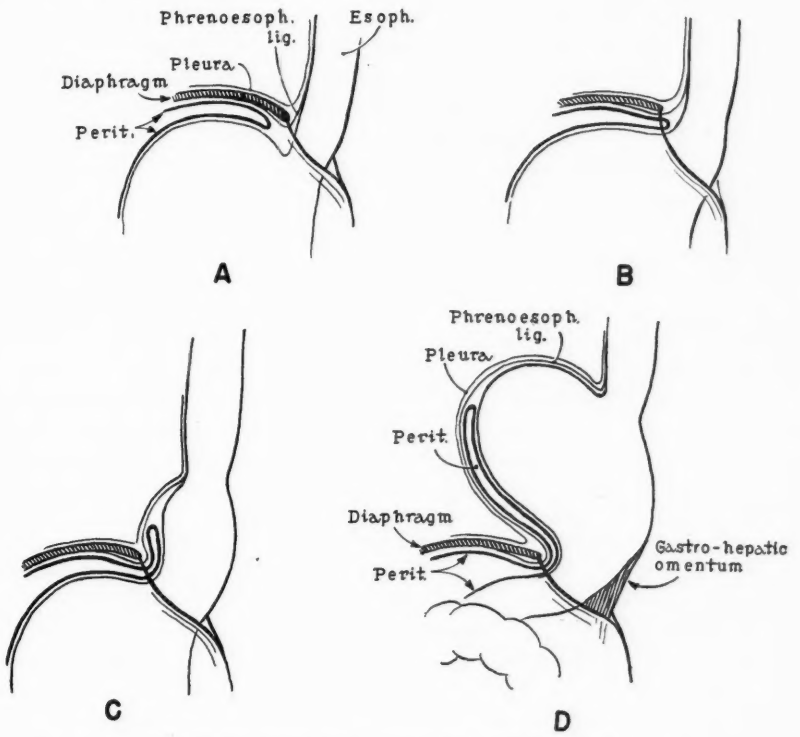


Fig. 2.—Diagram of the hiatus in the plane indicated in Fig. 1.

*A*, Normal. The hiatus lies almost vertically, with the esophagus passing obliquely forward into the stomach. The peritoneum is reflected onto the diaphragm anterior to the cardia. There is no peritoneum posterior to the cardia.

*B*, First stage of sliding hernia. The cardia is above the hiatus and the esophagus has lost its acute angle of entrance into the stomach. The hiatus is enlarged by spreading of the posterior crural fibers. The anterior peritoneal fold has been drawn backward and is just at the hiatus, but is not actually in the thorax.

*C*, Second stage of sliding hernia. The esophagus has shortened and the cardia is well above the diaphragm. The hiatus has enlarged both anteriorly and posteriorly and a fold of peritoneum has entered the chest, rolling back from the stomach as the latter rises.

*D*, Advanced stage of sliding hernia. The gastrohepatic vessels, though stretched, tend to hold the posteromedial, lesser curvature aspect, but further anterior hiatal stretching has allowed the fundus to roll upward to a position higher than the cardia carrying a large fold of peritoneal "sac" with it. Ultimately gastrocolic omentum or colon may pass upward into this "sac."

True congenital short esophagus hiatal hernia is rare. Only one case is included in this series. It is distinguished from acquired hernias by the presence of a direct segmental arterial blood supply from the intrathoracic aorta to the intrathoracic stomach.[3, 20, 28]

SYMPTOMATOLOGY

The characteristic symptoms of hiatal hernia of the sliding type are related primarily to reflux of stomach contents into the esophagus. They consist of heartburn, substernal and epigastric pain, a pulling sensation in the throat and episodes of sudden choking especially when recumbent at night. In severe cases the patient may find sleep possible only in a chair. Bending forward or stooping causes severe distress. If secondary esophagitis is present, dysphagia may be extreme, or hematemesis may be the presenting symptom. Extreme aerophagia and belching are frequent. The symptoms are often confused with those of cholecystitis and angina and, when these can be excluded, they may be attributed to "bolus hystericus." These unfortunate patients often suffer for many years, seeking help from many doctors and developing an overlay of functional symptoms that obscures the true cause of their difficulty more and more. Chronic esophagitis with or without actual peptic ulceration which then occurs may present additional difficulties both in diagnosis and treatment. In the ordinary case vomiting is rare, and the patient is usually obese, belying her complaints of difficulty in eating or "indigestion." It should be emphasized that these symptoms are unrelated to the size of the hernia.

In paraesophageal hernia, on the other hand, dysphagia and digestive symptoms are less frequent because the cardia is competent. It seems paradoxical that many patients with these sometimes huge herniations are relatively free of symptoms, whereas those with apparently insignificant or "incidental" sliding hernias may be almost incapacitated. When paraesophageal hernias do cause symptoms it is often due to pressure on the heart or lungs. These hernias occasionally become incarcerated with acute gastric obstruction and enormous dilatation, a complication which never occurs in sliding hernias. On the other hand, acute hemorrhage is rare.

The true congenital hernias may result in intractable vomiting in infancy. When this is not the case, later symptoms due to gastric reflux may occur as in sliding hernia, so that in adults the differential diagnosis may be impossible until operation.

*Indications for Operation.*—When extreme symptoms of obstruction, hemorrhage, or progressive incapacitating dysphagia are present there is little question that operation is necessary. But it may be hoped that with increasing understanding of the earlier symptoms and the safety and efficacy of repair, fewer patients will be allowed to reach this stage. There is no question that if suggestive symptoms occur in the presence of a radiologically demonstrable hernia and in the absence of any other lesion which might be responsible, operation should be done. Many small hernias, however, apparently cause little or no symptoms. At present it is not generally accepted that these should be repaired, but it is not known how many may eventually become symptomatic. The time may come when the mere presence of such a hernia will be sufficient indication for repair, in the absence of definite contraindications, as is now the case with inguinal hernia.

## OPERATIVE TECHNIQUE

The following technique was devised in 1949 with these principles in mind: Since the majority of hiatal hernias are of the simple sliding type in which the chief problem is a weakening and widening of the posteromedial crural fibers, allowing the cardia to slide upward into the mediastinum, and since the symptoms are primarily due to the resulting incompetence of the cardia, the main problem is to return the cardia to its normal position, hold it there, restore the esophagogastric angle, and bring together the posterior fibers of the right crus, reducing the hiatus to normal size. Since there is no true sac, there is no need to enter the peritoneal cavity. Recurrence should best be prevented by establishing firm union between the wall of the stomach all around the cardia and the adjacent undersurface of the diaphragm above and medial to the peritoneal reflection. Tight closure of the hiatus around the esophagus is unnecessary, may cause dysphagia, and is ineffectual since sutures in the muscular wall of the esophagus do not hold well. The phrenoesophageal ligament is often so thinned out as to be ineffectual as a structure with which to establish a firm repair. Phrenic nerve paralysis is unnecessary and probably harmful.[22]

Modifications of the basic technique may be necessary in dealing with large hernias, especially those containing colon or other viscera; in these, the peritoneal cavity may have to be opened, either through the sac or through a separate opening in the diaphragm. This is often true of the composite type, and always true of paraesophageal hernias. A thoracoabdominal approach is rarely necessary. The abdominal approach should be reserved for patients in whom intra-abdominal disease is suspected.

The posterior mediastinum is approached through a left posterolateral oblique incision over the eighth interspace. The latissimus dorsi muscle is divided, the serratus muscle retracted forward, and the paraspinal muscles retracted posteriorly after dividing attachments to the seventh, eighth, and ninth ribs. Depending on the habitus of the patient, the chest is entered through the eighth interspace, or, after resecting eighth or ninth ribs, through the rib bed. Division of the neck of one or both adjacent ribs may be necessary.

The diaphragm is retracted inferiorly and the pulmonary ligament is divided from the diaphragm to the inferior pulmonary vein. The lung is then retracted superiorly, and the cut edges of the mediastinal pleura, resulting from division of the pulmonary ligament, are retracted anteriorly and posteriorly by stay sutures. At the diaphragmatic hiatus the pleural incision is continued posteriorly across the aorta. The muscular crural fibers coming up from the origin of the right crus and passing between the herniated stomach and the aorta now come into view. They may be much attenuated or altogether absent (aorticohiatal hernia) but usually are a firm well-defined band. Anteriorly the pleural incision is also extended from the lower end of the pulmonary ligament toward the reflection of the pericardium. The crural fibers lying

medial to the esophageal hiatus now are seen, but full exposure may be difficult until the herniated stomach is retracted laterally. This is accomplished by lifting the esophagus above the cardia out of the mediastinum, passing a soft rubber tube around it for gentle traction, and then carrying the dissection downward over the herniated stomach to the hiatus. Special care should be taken to separate the stomach posteriorly from the aorta and to avoid injury to the vagus nerves. In true short-esophagus hiatal hernias, there may be segmental arteries arising from the aorta and passing to the gastric fundus. This is not true of the much commoner sliding hernia.

When the herniated stomach has been completely freed of its attachments in the mediastinum, it can be lifted forward and medially to expose the acute

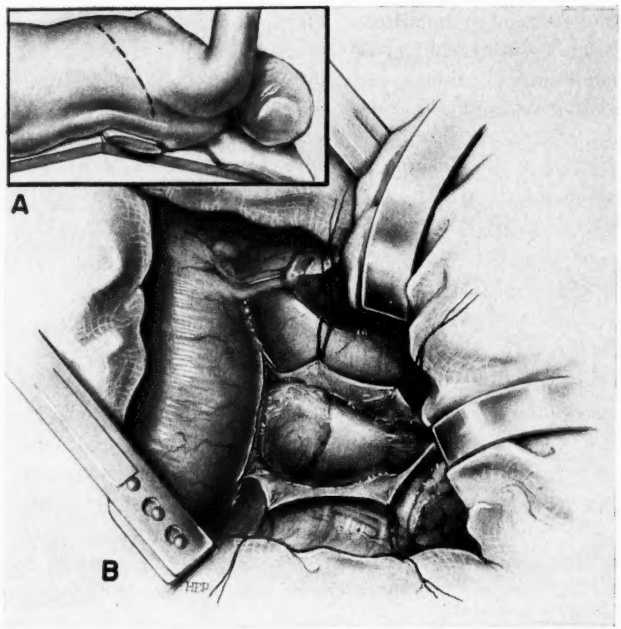


Fig. 3.—View of the usual situation found at operation for sliding hiatal hernia through left posterolateral thoracic approach (inset). The pulmonary ligament has been divided, the lung retracted upward, and the pleural edge retracted by stay sutures. The cardioesophageal junction is uppermost, and the herniated stomach carries with it a fold of peritoneum anteriorly.

angle of the posterior end of the esophageal hiatus, which is formed by overlapping sheets of the right crural fibers. This angle must be partially closed, and at the same time the esophagogastric junction must be fixed below it. Before beginning the repair, however, the anterolateral aspect of the herniated stomach must be separated from the undersurface of the diaphragm. Usually, but not always, a fold of subdiaphragmatic peritoneum has been rolled upward into the chest on this gastric surface as a "sac." This peritoneum can readily

be pushed back under the diaphragm without opening it. Stay sutures are placed in the muscular edge of the hiatus so as to hold it away from the stomach (Figs. 3 and 4).

The repair is now begun by placing 2 or 3 mattress sutures of 2-0 silk between the medial and lateral fibers of the right crus posteromedially in such a manner that the normal overlap will be restored when they are tied. The uppermost of these includes a firm "bite" of the muscular wall of the stomach just at the esophagogastric junction posteromedially. When this is tied, later, it will bring the cardia just below the united crural fibers, well down under the almost vertical portion of the diaphragm, thus restoring its obliquity. Mattress sutures of 2-0 silk are now passed through the diaphragm about 3 cm. from its hiatal margin, passing down from its pleural surface, then out between the diaphragm and herniated stomach but above the peritoneal fold, then up to

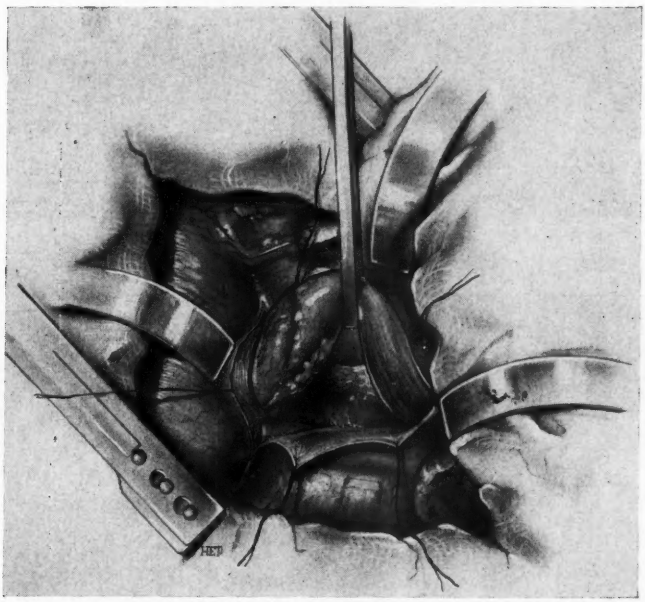

Fig. 4.—The esophagus and herniated stomach have been separated from their mediastinal attachments and retracted forward. A fold of peritoneum is attached to the anterior surface of the stomach; posteriorly there is attached loose tissue of the gastrohepatic omentum containing branches of the left gastric artery, which should not be injured. A stay suture is shown on the edge of the hiatus; usually several are used.

include a "bite" of the muscular wall of the stomach at a distance from the esophagogastric junction corresponding to the distance of the stitch from the margin of the hiatus, and then back under the diaphragm and up through its pleural surface a few millimeters from the point of beginning (Fig. 5). Usually 5 or 6 mattress sutures placed circumferentially around the hiatus are sufficient. If an asymmetrical thinning of the circumhiatal muscle is present,

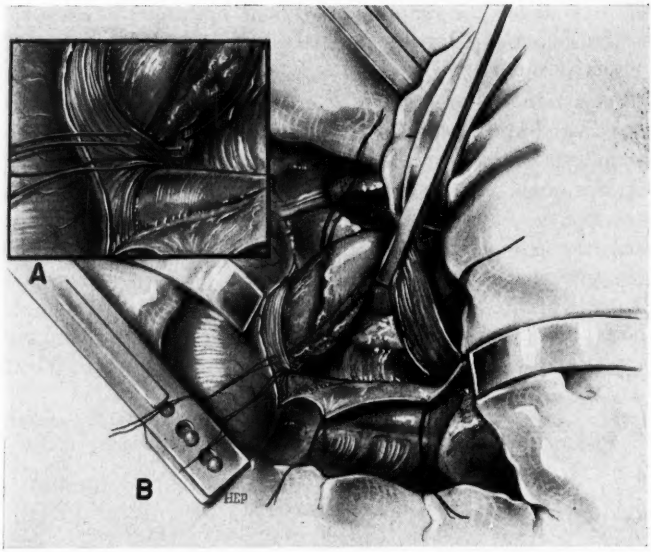


Fig. 5.—Placement of sutures for repair. The repair is begun posteriorly to restore the overlap of the right crural fibers and bring the cardia below them (inset). Mattress sutures are then placed circumferentially through the diaphragm and the stomach wall above the peritoneal reflection. Corresponding sutures to those shown are placed medially.

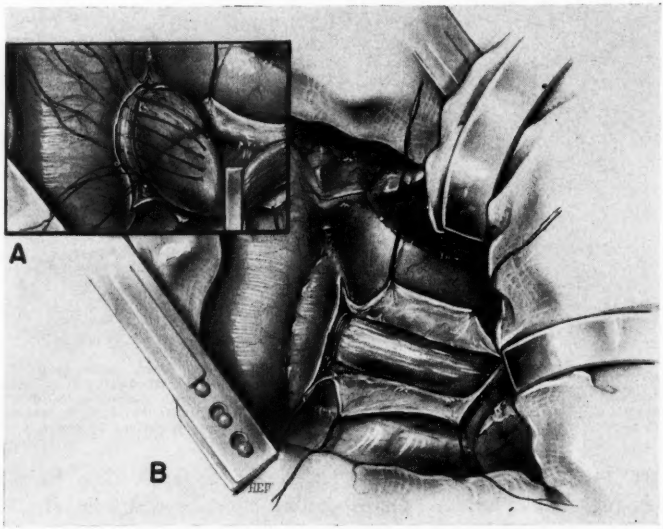


Fig. 6.—The repair completed. The tied mattress sutures reduce the stomach and create a cuff of diaphragm over the cardia. The crural sutures disappear behind the aorta. The hiatal edge is sutured to the fascial envelope of the esophagus without tension, as a final step.

most often in the medial crus,[18] the mattress suture in this region may be so placed as to restore the normal symmetry of the hiatus. In rare instances additional sutures may be needed in the diaphragm itself.

All sutures are placed before any are tied, the stomach remaining in its herniated position. The crural sutures are tied first, while gentle traction is applied to the circumferential mattress sutures, thus drawing the stomach down

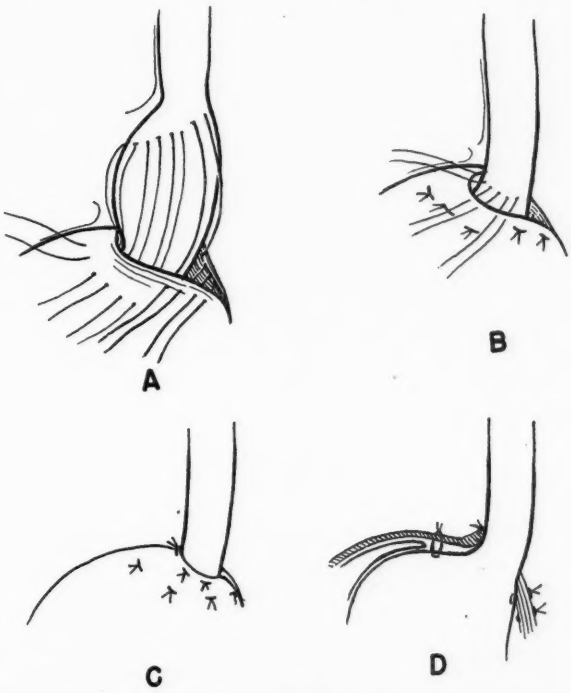


Fig. 7.—Diagram of method of repair of sliding hernia.

*A*, Mattress sutures have been placed; one between the separated crural fibers; one between the crural fibers including the cardia; and circumferentially through the diaphragm and the stomach wall. Corresponding mattress sutures are also placed medially.

*B*, The mattress sutures have been tied, drawing the stomach into anatomic position below the diaphragm and fastening it there. The angle of the cardia is restored. Sutures are placed between the edge of the hiatus and the fascial envelope of the esophagus.

*C*, The repair completed.

*D*, Diagrammatic section to show restoration of normal anatomy, with broad surface of attachment extraperitoneally anterior to the hiatus.

into anatomic position under the diaphragm. The mattress sutures are then tied. The stomach now lies normally, and a lax cuff of diaphragm is created around the esophagus. The edge of the hiatus is now sutured to the fascial envelope of the esophagus without tension, using fine silk (Fig. 6). The purpose of this suture is to ensure a broad surface of healing between the stomach wall and the diaphragm, rather than to hold the esophagus down or to close the hiatus around it (Fig. 7).

The chest is closed with fine silk and without drainage after repair of the pulmonary ligament over the esophagus and re-inflation of the lung. A naso-gastric tube is usually left in place from 12 to 24 hours in order to avoid gastric distention from air swallowing, after which the patient is allowed up and given a normal diet.

## STATISTICAL RESULTS

A total of 97 operations for hiatal hernias were performed on 96 patients (one recurrence having been repaired a second time) between Jan. 1, 1940, and Dec. 31, 1954, at the Presbyterian Hospital in New York. This period was chosen because, before 1940, the number of such operations was very small and there was no consistent theory of technical management. Actually the period covers the transitional one from an attitude of confusion and reluctance on the part of the surgeon, to one of relative understanding. Since it is evident that the end result of operation on this hernia, as on any other, is not certain for at least 2 years after operation, and since few of the reported series have concentrated on the long-term results of the operations described, it was decided to include no patients with a follow-up period of less than 2 years. It will be shown that during the last 5 years of the study period a method has been used in the commoner sliding hernia, which in our hands is more successful than any other. In the period subsequent to that studied, this method has been used with increasing frequency with what seem to be even better results, but it is felt that the postoperative period is not sufficiently long to be certain.

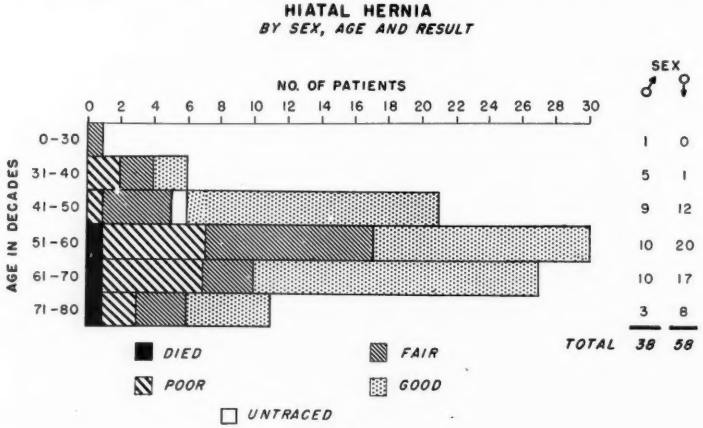


Fig. 8.

*Age and Sex.*—It is common knowledge that hiatal hernia is more common in the sixth decade of life, and commoner in women than in men. This series is no exception. It is interesting to note, however, that the proportion changes with age. In the fourth decade it is more frequent in men; in the fifth the numbers are approximately equal, and only in the sixth does the proportion shift to a 2:1 preponderance of women. This proportion is also the case in the seventh decade, and over that it is even greater. One would expect that results

would be better in the younger group and mortality greater in the older but this does not appear to be the case. Significant differences might be more evident in a larger series (Fig. 8).

*Operation Type.*—The operations were performed by a total of 28 different surgeons, using a great variety of techniques, especially in the earlier period. In general, the abdominal approach has been reserved for patients in whom it was uncertain that the symptoms were due to the hernia and in which abdominal disease was suspected. The great majority, even in the earlier period, were done through the left transpleural approach, and each of the more modern procedures have been tried in a number of cases, including, at first, that of Merendino, Varco, and Wangensteen,[19] and subsequently those of Allison,[2] Sweet,[27] and Lam.[15] In addition many variations were tried, according to the presenting problem and the surgeon's ingenuity. Analysis reveals no consistent pattern of success or failure in this group except that it becomes clear that the methods described in the literature are less successful when performed by surgeons who are interpreting what they have read, than when performed by the men who devised them. The method described here was devised at about the same time and is readily learned, depends on similar principles, and is effective in the usual sliding hernia (Fig. 9).

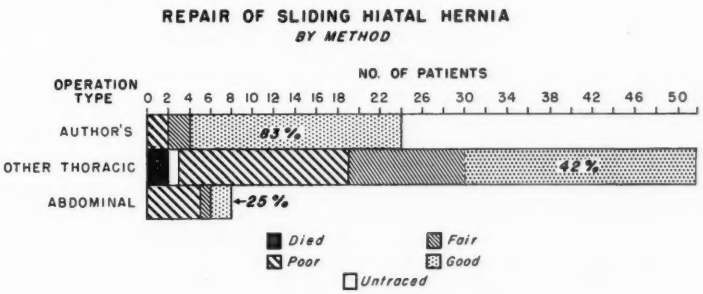


Fig. 9.

*Hernia Type.*—The great majority of the hiatal hernias in this series were of the sliding variety, 88 of the 97 operations being performed for this type (90 per cent). There were 6 paraesophageal hernias, 2 esophago-aortal hernias, and one infant with a congenital undescended stomach. All but one patient, who was untraced after 6 months, were followed for from 2 to 9 years after operation. The results are classified as "good" only if the patient is free of all symptoms of hiatal hernia and free of evidence of recurrence by x-ray study when that has been done. Sixty-five per cent of the patients had postoperative roentgenograms, 45 per cent of them made one year or more after the operation. If symptoms persist to a minor degree or if a small recurrence is seen by x-ray study with or without symptoms, the result is classified as "fair." If severe symptoms continue, whether or not recurrence is present, or if a sizeable recurrence is demonstrated, whether or not it gives symptoms, the result is classified as "poor." The over-all statistics are shown in Table I.

TABLE I. RESULTS OF OPERATION BY TYPE

| TYPE | GOOD | FAIR | POOR | UNTRACED | DIED | TOTAL | PER CENT GOOD |
|---|---|---|---|---|---|---|---|
| Sliding | 44 | 16 | 24 | 1 | 3 | 88 | 50 |
| Paraesophageal | 3 | 2 | 1 | | | 6 | 50 |
| Esophago-aortal | | 1 | 1 | | | 2 | 0 |
| Congenital | | 1 | | | | 1 | |
| All types | 47 | 20 | 26 | 1 | 3 | 97 | 48 |

*Sliding Hernia.*—The results have been tabulated by result and method in Table II. All of the deaths were in this group. One patient died of shock on the first postoperative day following a total gastrectomy through a thoraco-abdominal approach for acute hemorrhage from the stomach. No source of bleeding was found in the resected stomach or at autopsy. In retrospect it seems likely that simple repair of the hernia might have been better judgment, since diffuse bleeding from the congested intrathoracic pouch is a well-known complication of hiatal hernia. One patient died on the third day after operation with evidence of progressive brain damage probably secondary to a period of anoxia during operation. The third suffered an acute mesenteric arterial embolus on the third day after operation. The heart was fibrillating and the patient had had previous emboli.

TABLE II. RESULTS OF OPERATION ON SLIDING HERNIA BY METHOD

| METHOD | GOOD | FAIR | POOR | UNTRACED | DIED | TOTAL | PER CENT GOOD |
|---|---|---|---|---|---|---|---|
| Author's | 20 | 2 | 2 | | | 24 | 83 |
| Other thoracic | 22 | 11 | 16 | 1 | 2 | 52 | 42 |
| Abdominal | 2 | 1 | 5 | | | 8 | 25 |
| No repair | | 2 | 1 | | 1 | 4 | 0 |
| All methods | 44 | 16 | 24 | 1 | 3 | 88 | 50 |

The 24 poor results fall into 4 main groups. Operations done primarily for anginal pain failed to relieve the angina in 4 patients. Six patients with severe long-standing chronic esophagitis were not relieved by repair of the hernia, including the 2 poor results following repair by the method described here. Presumably some form of resection is a more appropriate method of treatment in this group. Large recurrences were responsible in 7 cases. The remaining 7 continued to have symptoms despite apparently successful repair. One of these died of carcinoma of the pancreas within the year, and a second was operated upon for carcinoma of the rectum 4 months after repair of the hernia, dying of recurrent carcinoma 5 years later. These must be classified as missed diagnoses, but it is interesting to note that the man with rectal carcinoma was operated upon through an abdominal approach and the abdomen was explored. The other 5 were severe psychoneurotics, whose continued complaints appeared to have no organic basis. One may question whether these poor results were not related to long neglect of the patient's hiatal hernia symptoms no less than those with chronic esophagitis.

*Paraesophageal Hernia.*—The 6 patients in this group are especially interesting. One suffered from mild upper abdominal distress for many years before an acute episode of pain and intractable vomiting brought her to emergency operation. Through an upper abdominal incision, an incarceration of almost the entire stomach was reduced. The hernia was not repaired and she has remained well, with the same symptoms as before incarceration for the ensuing 6 years. Three others had a history of previous episodes of acute pain and vomiting suggesting transitory incarcerations; one had many of increasing severity, the others each had only one previous episode, 10 and 4 months before operation, without any previous symptoms. Two of these were also operated upon as emergencies. One, after 5 days of acute symptoms, was treated "conservatively" for 8 days before operation due mainly to his physician's unfamiliarity with the syndrome. There was gangrene of the omentum and extensive inflammatory exudate and adhesions were present over the incarcerated stomach. A thoracoabdominal incision was required to achieve reduction. His stomach remained obstructed, however, until 11 days later when the abdomen was reopened and the stomach found held in an acutely kinked omega loop by adhesions. Evidently the original reduction had been "en masse." The third patient to have an emergency operation for incarceration was recognized promptly and operated upon only one day after the onset of symptoms. There were no adhesions or gangrene, the hernia was reduced and repaired through a thoracic approach with diaphragmatic incision, and he made a prompt recovery. This was the patient who had had many previous attacks. He has been completely well for 4 years, with no abnormality by x-ray study. The fourth patient had her first attack one month before operation but was not acutely incarcerated. She was operated upon through the abdomen but the incision had to be extended into the chest to accomplish reduction. Repair was from the abdominal side. For nearly 3 years she was free of symptoms; then, following a fall with a blow on the abdomen, the whole symptom complex returned acutely. She was reoperated through the thorax in another hospital, but in the ensuing 2 years continues to have mild symptoms suggesting recurrence. The other 2 patients had no gastric symptoms, but were operated upon because of cardiac difficulties. One suffered from palpitation and substernal distress, the other frank angina after eating or when recumbent. Both were repaired transthoracically with incision of the diaphragm, and both have remained free of hernia symptoms for over 2 and over 5 years, respectively.

It is evident, then, that paraesophageal hernias, though usually larger than the sliding type, may be less troublesome unless they cause pressure on the heart or become incarcerated. The latter complication should be recognized and dealt with at once, but it is preferable to correct the condition electively, through a thoracic approach, with incision of the diaphragm to ensure complete reduction.

*Esophago-Aortal Hernia.*—There were 2 examples of this interesting anatomic variant. The first was in a woman of 57 with life-long mild symptoms. It was found that the aorta was actually displaced in a wide arc around the intrathoracic stomach, suggesting a congenital origin. The gastric blood supply,

however, was normal. A repair was done according to the method of Merendino, Varco, and Wangensteen.[19] She remained free of recurrence by x-ray examination but continued to have moderate dysphagia due presumably to tight closure of the diaphragm around the esophagus. Four years after operation she died of acute coronary occlusion. The other was in a woman of 55 years of age who had suffered from postprandial and decubitus substernal pain for years and who presented the symptoms of acute hematemesis and melena. The aorta was not distorted but the right crural fibers between it and the stomach were absent. This may, therefore, have represented simply a variation of an ordinary sliding hernia in which the left leaf of the right crus had atrophied completely. A repair was done by drawing the left crus medially across the aorta and suturing it to the right after reducing the hernia. It is evident that this is unanatomic and the probability that too much tension was required is indicated by the fact that it had recurred 8 months later. A second operation was done in another institution, the details of which are unknown, but she still has her original symptoms 9 years later.

*Congenital Hernia.*—The single infant in this series suffered from increasing vomiting and inability to take solid food for the 18 months between birth and operation. The esophagogastric junction was at the level of the pulmonary hilus, and the intrathoracic stomach was supplied by segmental arteries from the aorta. This, therefore, represents failure of the stomach to descend to its normal position in fetal life, rather than a true hernia. Although often termed "congenital short-esophagus hernia," in this case the esophagus seemed contracted rather than short, and the stomach could readily be replaced below the diaphragm when its attachments to the aorta had been divided. Repair was by the method of Merendino, Varco, and Wangensteen.[19] Two years later he still has difficulty in swallowing solid foods due to too tight closure of the diaphragm around the esophagus as demonstrated by x-ray study. It seems probable that further surgical correction will be needed, since a secondary megaesophagus is developing.

## DISCUSSION

These results show that many past failures of repair of hiatal hernia are the result, as emphasized by Lam,[15] of incomplete understanding of the anatomy and physiology of the hiatus. Mere narrowing of the hiatal orifice and attempts to resect the "sac" and fasten the esophagus to the hiatal rim is inadequate because it does not restore the esophagogastric angle, permitting continued gastric reflux. If the hiatus is closed tightly around the esophagus, the latter will be unable to expand properly to admit a swallowed bolus and dysphagia may be worse than before. This is the objection to earlier procedures such as that described by Merendino, Varco, and Wangensteen,[19] though this may still be used in exceptional cases in which the esophagus has lost its resilience due to prolonged inflammation and fibrosis. Such late cases and those complicated by deep peptic ulceration are better treated by resection and transplantation of a segment of jejunum or ileum as advocated by Barrett.[3] In these, the vagus

nerves are necessarily divided and simultaneous pyloroplasty is advisable but gastric resection is not necessary unless gastric or duodenal peptic ulcer is also present.

The procedure described in this paper is carried out, in the usual sliding hernia, entirely through the chest without opening the peritoneal cavity. It is felt that this is simpler and carries less morbidity than when the diaphragm is opened as advocated by Allison[2] and Lam,[15] though the basic principle is the same. Sweet[27] also finds it usually unnecessary to open the diaphragm, but his plication of the sac seems to us a less secure fixation of the stomach beneath the diaphragm. The transabdominal methods of Harrington[12] and Beardsley[4] secure the stomach, but are more difficult technically and carry a higher risk. We do not agree with Tanner[29] that the diagnosis is easier to verify from below. Obviously if intra-abdominal disease is responsible for the patient's symptoms the abdomen should be explored. It is axiomatic in the treatment of inguinal hernia that the presence of the hernia should not distract the surgeon from treatment of more serious disease in the abdomen, and this is even more true of hiatal hernia.

The differential diagnosis between hiatal hernia symptoms and true angina pectoris may be very difficult. In some cases it appears that the hernia may act as a "trigger" mechanism, but it is noteworthy that, where evidence of coronary disease and hiatal hernia coexisted in this series, repair of the hernia was ineffectual in relieving symptoms. On the other hand when gall bladder disease is also present, or in the syndrome called "Saint's triad," the hernia is most often the chief offender, as brought out by Palmer.[21]

It is evident that the decision to operate depends above all on an accurate evaluation of symptoms. This is often a difficult task especially in those patients who have developed, through years of medical misunderstanding, a serious overlay of functional complaints. Although it may be true that many small hernias of the hiatus cause minimal symptoms, and in many others the symptoms can be relieved by simple measures such as antacids, posture, and weight reduction, we agree with Blades[5] and Graham[10] that the time is past when a hiatal hernia can be considered an incidental finding. It has been shown by Sprafka, Azad, and Baronofsky[25] that a large proportion of these hernias progress over the years and ultimately develop serious complications. It is agreed by all surgeons of experience that patients with serious complications require more radical and more dangerous operations, and that the results are less satisfactory than if they had been operated upon earlier. There is even reason to believe that the incidence of carcinoma of the esophagus is definitely higher in this group. Therefore we cannot agree with the prevailing opinion, as expressed by Boyd,[6] Harrington,[12] Souter,[24] Stensrud,[26] Sweet,[27] and Tanner[29] that these hernias should always be treated nonsurgically at first, and that only those patients with marked and obvious symptoms should be operated upon. Rather it would seem that, as is the case in inguinal hernia, simple, effective

and safe methods are available for early uncomplicated hiatal hernias, and that in the absence of other disease or contraindications, the earlier they are repaired the better.

## SUMMARY AND CONCLUSIONS

1. The present status of our understanding of the anatomy, physiology, and symptomatology of hernias through the esophageal hiatus of the diaphragm is reviewed.

2. The current classification of such hernias is presented.

3. A method for repair of uncomplicated sliding hiatal hernia is described which has been used for 8 years.

4. The long-term results, from 2 to 9 years after operation in a series of 97 operations in 96 patients with hiatal hernia of various types have been reviewed. A variety of procedures were used by 28 surgeons in one hospital.

5. The results show:

a. The great majority of hiatal hernias are of the sliding type. There were 87 sliding, 6 paraesophageal, and 2 esophago-aortal hernias. One infant is included with an intrathoracic stomach presumably due to congenital failure of descent.

b. The operation described was carried out in 24 of the 88 operations for sliding hernia. Results were good in 83 per cent. Other transthoracic methods were used in 52 operations with good results in 42 per cent. Transabdominal operations were reserved for complicated cases and were done 8 times, with only 2 good results (25 per cent).

c. In 4 cases the hernia was not repaired at operation. One of these and 2 other patients with sliding hernia died as a result of operation, none of them following the described procedure.

d. Paraesophageal and esophago-aortal hernias require more complex operations, though the same principles apply. Three or 50 per cent of the patients with paraesophageal hernias were operated upon for acute incarceration, 2 with good and one with fair results. Only one of the elective repairs ended with a good result. There were not good results in repair of either of the esophago-aortal hernias and the result in the congenital hernia was only fair. There were no deaths in this group.

6. It is concluded that the method described, embodying principles similar to other methods reported recently by others, is effective in a high proportion of uncomplicated cases of sliding hernia and that early operation is therefore indicated.

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*(For Discussion, see page 774.)*

# AN OPERATION FOR HIATUS HERNIA WITH SHORT ESOPHAGUS

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When a patient has an irreducible hiatus hernia of the sliding type, with an incompetent hiatus and a short esophagus, his symptoms are usually very severe. This is in contrast to the same state of affairs in a patient with good crural muscle at the hiatus. This last group is often referred to as having a gastric-lined esophagus which is compatible with a comfortable life and few symptoms. In infants and young children, the condition frequently develops from one to the other. The infant has poor control over most of its sphincters and the mechanism at the lower end of the esophagus may be involved in this way. As months pass the muscle fibers of the right crus gain strength and gastric reflux is controlled. This may be associated with complete reduction of a previously existing, reducible, sliding hiatus hernia but, in some cases, control of reflux is developed at the esophageal hiatus in spite of the absence of complete reduction of the sliding hiatus hernia. In such cases the hernia has become irreducible, but reflux from the main body of the stomach into the gastric tissue within the chest has come under control. It is these patients which are often referred to as having a gastric-lined esophagus and in whom symptoms are often slight. There is a small group of young patients in whom neither reduction of the hiatus hernia nor control of the reflux occurs. This group together with a larger one made up of patients who develop the same condition in later life form the type of case now under consideration.

Most of the operations which have been suggested for the relief of this condition are large and ill-suited to patients who are frail and often aged. It is also to be noted that they usually leave the patient with a very disorganized alimentary tract so that digestion is neither effective nor comfortable. Dissatisfaction with the situation this presents had lead many surgeons to return to a palliative line, and to treat such patients by posture and dilatation of the lower end of the esophagus if dysphagia is a problem.

It is not disputed that there is a place for such treatment but, in many cases, it does not give relief and always must be regarded to some extent as an admission of defeat.

What is required is an operation which does not disorganize the patient's digestive apparatus too much and which is reasonably easily tolerated by even a frail and aged person. A lead in the direction of how this might be effected was developed from the refashioning of the author's standard operation for

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hiatus hernia in the period 1952 to 1953. The remodelled operation was produced as a result of anatomic and physiologic investigations which were recorded in *Thorax* in 1954. This work had been started following a general discussion of hiatus hernia and the operative results at the annual meeting of the Society of Thoracic Surgeons of Great Britain and Ireland in 1951. From the papers and discussion which then took place, it was clear that failure to control reflux was common and that there was a high incidence of actual recurrence of the hernia.

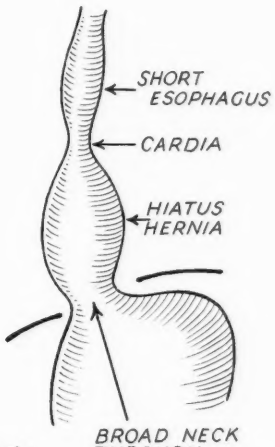


FIG. 1.

Fig. 1.—This is a diagrammatic representation of a hiatus hernia and short esophagus. A broad neck of gastric tissue passes through the esophageal hiatus and there is no angle between the stomach above the diaphragm and that below. Free reflux is certain to take place from the body of the stomach into the pouch of the hiatus hernia and up the esophagus.

The investigations demonstrated that although many factors helped to control reflux, there was one which could be effective alone. This was the acute angle of implantation of the esophagus into the stomach which, in turn, is produced by normally functioning crural muscle. This muscle contracts with the rest of the diaphragm and maintains the angle of implantation of the esophagus into the stomach at all phases of the respiratory cycle. The absence of symptoms in children with the so-called gastric-lined esophagus is a demonstration of the efficacy of this control.

When the muscle of the right crus has become weak or stretched, the angle between the esophagus and the stomach is opened out and reflux occurs. The amount of reflux and the consequent symptoms are related to this and not to the size of the hiatus hernia which, in some cases, may not be present in spite of gross symptoms of reflux.

The refashioned operation sets out to control reflux by suturing the fibers of the right crus above and in front of the esophagus so as to lower the point at which the esophagus joins the stomach and so make the angle of junction more acute. The results have been satisfactory and it has been demonstrated

that reflux can even be controlled although some gastric tissue is left above the diaphragm. This is not surprising in view of the observed facts with the group of cases of so-called gastric-lined esophagus.

A technical problem was found in the cases where stomach tissue was left above the diaphragm because of the bulk of tissue which occupied the esophageal hiatus. This made the plastic operation of suturing the two halves of the right crus difficult and less effective. From this point it was a short step to devise a method of reducing the size of the intrathoracic pouch so that the amount of gastric tissue left in the chest was as small as possible so that only a narrow tube of gastric tissue passed through the esophageal hiatus.

It may still be argued that gastric tissue should not be left above the diaphragm and used in this way as a connecting link between the esophagus and the main body of the stomach, but there is evidence from various sources to show that it is a reasonable line. First, it gives rise to little trouble in the children with gastric-lined esophagi; second, the amount of tissue is reduced to the minimum in the suggested operative technique; and third, the gastric tissue left is relatively innocuous. Berger[1] demonstrated that no hydrochloric acid was produced adjacent to the cardia while Billenkamp[2] showed that the area of the magenstrasse was devoid of peptic glands.

## GENERAL PLAN OF OPERATION

In view of the experience from congenital defects in this area and the work of Berger and Billenkamp, it seems reasonable to put forward a plan for using a gastric connecting link between the short esophagus above and the main body of the stomach below the diaphragm, provided certain precautions are taken.

First, it is necessary to use only gastric tissue of the magenstrasse. Second, an effective valve action must be obtained so that reflux is prevented from the main body of the stomach into the connecting link and esophagus above. Third, it seems desirable that as narrow a tube of stomach as possible is used so that the amount of gastric secretion should be minimal.

## TECHNIQUE OF OPERATION

The patient is placed on his right side with his whole body rotated a little backward. An abdominothoracic incision is made from the umbilicus to the edge of the trapezius along the line of the seventh or eighth ribs. The chest and abdomen are opened by resection of one or other of these ribs. It is found desirable to remove a segment of the costal margin. The incision in the diaphragm should be made as far laterally as possible so as to minimize the number of filaments of the phrenic nerve which are cut. For the same reason, it should only be taken to a point ½ inch short of the central tendon. This is important to have generally good function in the diaphragm after this operation. The small amount of lateral weakness is not a disadvantage.

The mediastinum is dissected so as to demonstrate the short esophagus and stomach pouch. In some cases it will be found that so much extra length of esophagus can be obtained by this dissection that a standard plastic repair can be done. Providing this is not the case, the gastric pouch is carefully

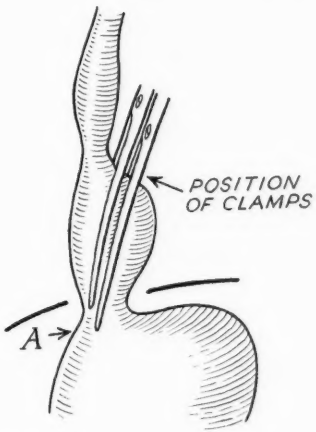


FIG. 2.

Fig. 2.—At this stage in the operation all the vascular attachments would have been cleared from the herniated portion of stomach. This freeing would have been carried below the diaphragm and the gastrosplenic vessels would have been divided. A further portion of the stomach is then drawn up into the chest before applying the clamps as shown. The points of the clamps are brought as close as possible to the lesser curve at *A*.

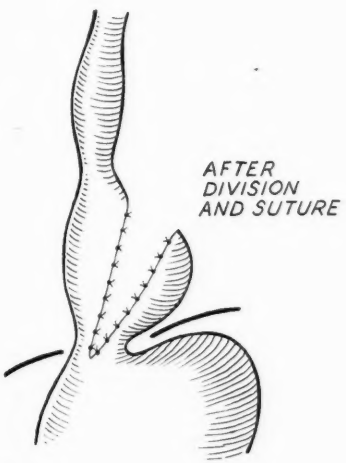


FIG. 3.

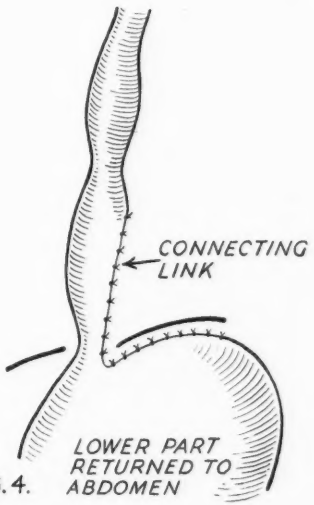


FIG. 4.

Fig. 3.—The stomach has now been incised between the clamps. The part adjacent to the lesser curve is to form the connecting tube. The part to the patient's left is to be returned to the fundus.

Fig. 4.—The connecting tube only now remains above the diaphragm. The lateral part of the stomach pouch has been returned below the diaphragm and increases the size of the fundus. An acute angle of implantation of the connecting tube into the stomach has been obtained.

cleared of vascular attachments so that its surface is smooth and clean. In order to do this it is useful to be able to inspect the stomach from below and to bring extra stomach up into the chest.

When this stage is completed, the anesthetist passes a No. 9 (English gauge) stomach tube from the mouth into the intra-abdominal part of the stomach. Parker Kerr clamps are then applied as shown in Fig. 2. The distal end of these clamps are brought as close as possible to the small stomach tube and lesser curve. The object of this is to make certain that the tube of stomach at this point will be as narrow as possible. The stomach is incised between the clamps and the free cuff sewn up before the clamps are removed.

After removing the clamps, the suture line is invaginated. The position now is as shown in Fig. 3. The part of the stomach pouch to the left is now returned below the diaphragm and has the effect of enlarging the fundus. Above the diaphragm a portion of the pouch remains which will from now on be referred to as the connecting tube.

The last phase of the operation is the plastic repair as described in *Thorax*, September, 1954. It is done from below the diaphragm. The under surface of the fibers of the right crus are dissected clean of everything except peritoneum which is carefully preserved. The limbs of the right crus are now sutured together above and in front of the connecting tube. This produces an acute angle between the connecting tube and main body of the stomach. As the diaphragm is closed the stomach is included in two of the stitches in order to retain the connecting tube and esophagus in position. The chest and abdomen are closed with intercostal drainage for 48 hours.

## RESULTS

The operation has been carried out on 11 patients. Nine were females and 2 were males. The ages were between 50 and 70 years in 9 of the patients. The other 2 were aged 31 and 37 years. All the patients had gross symptoms. Dysphagia was prominent in 8 patients and one had had many esophagoscopies for dilatation. At esophagoscopy, esophagitis was marked in all cases, with ulceration noted in 5. The esophageal length was between 29 and 34 cm. It is to be noted that the grossest forms of short esophagus have not been treated this way so far.

There was one postoperative death in a male patient of 67 who had a sudden collapse which was thought to be due to a coronary occlusion, on the fourth day after operation. One female patient, aged 37, developed a left subphrenic abscess with a leak from the suture line in the stomach. The whole condition subsided spontaneously with drainage of the pus. The remaining 9 made uneventful recoveries.

Of the 10 surviving patients, 2 are too recent for follow-up. The other 8 were all operated upon between April, 1955, and September, 1956. Of these, a satisfactory control of reflux was not obtained in the first patient, who was a female of 31. Although her symptoms are materially relieved, the result cannot be considered as really satisfactory. The postoperative result in the other 7 is excellent. A check, barium swallow has been done in all of these

patients between 8 and 12 months from the operation and in 6 no reflux could be produced. In one only, slight reflux occurred. At the same period after operation, these patients were also checked by esophagoscopy and complete absence of esophagitis and stricture was noted. No patient has required postoperative esophagoscopy and dilatation. This is in spite of the previous dilatations required in one case. It is to be noted that, in the immediate postoperative period, marked dysphagia was often present and this may remain a serious problem for 3 weeks with some persistent difficulty for several months. Some degree of immediate postoperative dysphagia is regarded as desirable and it is felt that if it is not present the operation has not been done properly.

The general improvement in these patients has been quite remarkable and in many cases their whole outlook has been altered. This is particularly noticeable in 2 of the patients, one of whom was also suffering from narcolepsy and the other from severe Parkinsonism. The symptoms from these latter conditions became much less troublesome after the operation.

## CONCLUSION

The operation which is suggested is of considerable magnitude but is still much smaller than esophagojejunostomy, and is well tolerated even by old and weak patients. It leaves the digestive arrangements near normal and there is no problem afterward with nutrition. Excellent late results have been obtained in 7 patients, but the unsatisfactory state in the first case must be taken as a warning of the need to narrow the connecting tube sufficiently so that a really good angle of implantation can be obtained. The procedure has not been used on the very short esophagi where the cardia is in the region of the aortic arch or above. However, the reason for this is only conservatism and it is felt that results justify its use for these also.

Attention has been drawn to the spontaneous relief of dysphagia after this operation. By this it is intended to suggest that many strictures which are generally believed to be fibrous are, in fact, mainly produced by reflex spasm. In these cases when the irritation of reflux has been removed and the esophagitis has subsided the dysphagia will no longer be present. While putting this forward as an observed fact, it is fully appreciated that, in some strictures, fibrous change is present and it is anticipated that esophagoscopy may well have to be done for dilatation in some cases in the future.

## SUMMARY

An operation is described for the condition of hiatus hernia with short esophagus.

The method involves the use of a small portion of stomach as a connecting link between the esophagus above and the main body of the stomach below the diaphragm.

The results of the use of the procedure in 11 patients is described.

## REFERENCES

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2. Billenkamp, H.: Beitr. path. Anat. **82**: 475, 1929.
3. Collis, J. L., Kelly, T. D., and Wiley, A. M.: Thorax **9**: 175, 1954.

## Discussion

(Papers by Humphreys, Ferrer, and Wiedel [page 749] and Collis [page 768])

DR. RICHARD H. SWEET, Boston, Mass.—These are two valuable papers. That by Mr. Collis proposing a new operation for a very difficult situation is interesting. In one respect it touches upon my own experience. For some time I have found it rather embarrassing to have observed so few cases of esophagitis after an esophagectomy with esophagogastric anastomosis when so many of my colleagues complain about its frequency. I wonder if this might not be the result of the fact that I have always made an effort to implant the esophagus into the wall of the stomach at an oblique angle, with the thought that this maneuver might eliminate some of the regurgitation of gastric juices.

About Dr. Humphreys' paper—this is one of the best presentations on the subject that I have heard. His diagrams are by far the best I have seen in presenting the actual anatomy of the situation, which is so important. His contribution to the technique by avoiding dealing with the fold of peritoneum, which he chooses not to call a sac but which I have always thought of as a sac because I regard this as a sliding hernia, is, I think, ingenious and valuable. Sometimes this enormous fold or sac of the peritoneum is a problem and I, along with Mr. Allison and others, have dealt with it by opening the diaphragm and often excising it. Dr. Humphreys' technique avoids that.

Without taking too much more time, I would like to have Dr. Humphreys comment, if he will, on how to make a diagnosis of recurrence by x-ray, because I find that in a number of instances the roentgenologist talks about small recurrences which I believe are probably explainable on the basis of the first paper we heard today where we saw it in the moving picture demonstration, as a reaching up or bulging up of the gastric mucosa into the cardiac region, which, if it is caught at just the right moment, might look like a recurrence which does not actually exist.

DR. ORVILLE F. GRIMES, San Francisco, Calif.—I should like to discuss Mr. Collis' paper. We have been interested in the problem of acquired short esophagus with stricture, not so much because of the number of cases, but rather because of the difficulties in management of these patients. The incidence of esophagitis following esophagogastrostomy after excision of the stenotic area certainly makes this operation less than desirable. In order to attack the problem directly, we set out to determine in the laboratory whether a naturally occurring valve in the gastrointestinal tract might not be substituted for either the narrowed lower esophagus or the excised cardioesophageal junction.

(slide) Working with Dr. John Najarian in the laboratory, the ileocolic valve was transposed from its natural habitat to the area of the cardioesophageal junction. The ileocolic artery divides near the cecum into an artery which progresses upward along the ascending colon and parallels the terminal ileum for a considerable distance. This anatomic arrangement exists not only in the animal but also in man.

This arterial pattern provides the opportunity of utilizing a pie-shaped segment of mesentery, including a short segment of the ascending colon, the cecum, and the segment of the distal ileum, varying in length from 15 to 25 cm. This segment can be rotated upon its apex with ease and carried up to the cardioesophageal area to replace the distal esophagus from the level of the inferior pulmonary vein to the cardioesophageal junction and beyond.

(slide) This shows how the entire segment, consisting of the ileum, cecum, and ascending colon, can be brought up in the dog through the omentum.

(slide) In the human being, the segment should be placed retrogastrically. An anastomosis between the ileum and the transected esophagus has been accomplished, and the anastomosis between the short segment of ascending colon and the gastric stump is about to be completed.

(slide) This shows the completed anastomosis in both areas and the ileocolic valve lying in the region of the previous cardioesophageal junction. A pyloroplasty has been done because the vagus nerves were divided to simulate the condition in the human being.

(slide) These animals were tested after a 6-week resting period, with daily injections of histamine in beeswax for 45 days. This slide shows the esophagus, the anastomosis be-

tween the esophagus and the distal ileum, the ileocolic valve, the short segment of the colon, the stomach, and the duodenum. As you can see by the arrows, under the stimulus of histamine three distinct ulcerations have occurred in the duodenum; some gastritis has occurred, but the colon segment, the ileal segment (protected by the ileocolic valve), and indeed the esophagus, remain free from inflammation.

(slide) This shows another such preparation: esophagus, terminal ileum, ileocolic valve, colon, and stomach. Here again an ulcer has been produced by histamine stimulation, but the normally functioning ileocolic valve has prevented both ileitis and esophagitis.

This procedure has been applied only to 2 human patients, one of whom did not survive the operation. I think, however, that this was due less to the magnitude of the procedure than to poor selection of the patient. The other patient has remained entirely well. I apologize for this meager experience, but hope that the presentation of this material will stimulate the members of this Association to evaluate this procedure as part of the surgical armamentarium in this problem.

DR. EARLE B. KAY, Cleveland, Ohio.—I would like to discuss Dr. Humphreys' very valuable paper with reference to the prevention of regurgitant esophagitis. My opinion is based on an experience of over 250 patients operated upon for hiatal hernia.

(slide) Most surgeons, including ourselves in the past, have made no attempt to correct the patulous esophagogastric stoma with its inadequate closing mechanism for preventing regurgitation of gastric juice into the esophagus which results when the herniated stomach and terminal esophagus is replaced "en masse" into the abdominal cavity. Occasional patients would be seen postoperatively complaining of the same symptoms of esophagitis as were noted preoperatively. Checkup barium roentgen examinations of the esophagus and stomach would not show any recurrence of the hiatal hernia but would show that the normal anatomic relationship of the esophagus entering the stomach at an angle on the lesser curvature of the stomach was lost and that, instead, it entered directly into the cardia. As noted in this slide, there is no stomach bubble and no closing mechanism at the esophagogastric junction.

(slide) For the past 3 years, in approximately 50 patients, I have attempted to correct this detail by suturing the cardia of the stomach to the left lateral wall of the esophagus for a distance of approximately 1½ inches so that the normal angulation between the esophagus and stomach would result. The fundus of the stomach would also be sutured to the dome of the diaphragm to be sure that during the healing process a position for a stomach bubble would be attained. When the esophagus has been shortened by esophagitis it is occasionally more difficult to perform this maneuver, but in the majority of instances it is possible. The other usual aspects of hiatal hernia repair are then followed.

(slide) Here is an x-ray following such a repair showing the entrance of the esophagus into the lesser curvature of the stomach and a large stomach bubble. Since such hernias have been repaired in this manner, there have been no instances of patients complaining of esophagitis postoperatively.

DR. DAVID H. WATERMAN, Knoxville, Tenn.—I was particularly intrigued by Mr. Collis' operation, as was Dr. Sweet, but I wish to direct my comments to Dr. Humphreys' excellent paper. I think Dr. Humphreys' emphasis on a herniorrhaphy that restores the normal anatomy is extremely important. For a number of years we tried the esophageal transplantation techniques of Merendino, Varco, and Wangensteen, and of Rives and Baker. The results were unsatisfactory in two respects; first, the recurrence rate was about 25 per cent and, second, the incidence of regurgitation and esophagitis was extremely high. Since that time we have been performing Mr. Allison's repair, and have had no recurrence and very little esophagitis in 37 consecutive cases.

Dr. Humphreys' operation is simpler than Mr. Allison's technique in that the diaphragm is not incised and phrenic nerve fibers thus severed. On the other hand, the diaphragmatic incision permits the suturing of the stomach to the diaphragm under direct vision, and affords the opportunity of exploring the upper abdomen. We feel these are often definite advantages.

I, too, am very much interested in how Dr. Humphreys diagnoses recurrence of the hernia. From a roentgenological standpoint I believe it is rather difficult.

DR. CONRAD R. LAM, Detroit, Mich.—I should like to compliment the work of Dr. Humphreys because it agrees so well with what Dr. Kenny and I presented at the San Francisco meeting several years ago. In the meantime we have had several recurrences, or at least what appeared to be recurrences, and second operations have been done. There may be a place for some additional steps in technique. The hiatus hernia is something which responds to modifications in the operation here and there, and the nice suturing of the stomach to the diaphragm which Dr. Humphreys showed may be important.

With regard to Mr. Collis' very ingenious operation, I think if I should ever encounter a short esophagus in an acquired hernia, I should wish to utilize his procedure. However, I have never seen a short esophagus in this situation.

DR. ROBERT H. WITMER, Lancaster, Pa.—I should like to ask Dr. Humphreys a question. Many of us have moved the hiatal opening nearer the dome of the diaphragm when we have encountered cases in which the esophagus is short, and we would like to know if this is the wrong thing to do.

DR. STUART W. HARRINGTON, Rochester, Minn.—President Haight, I appreciate your asking me to discuss these papers and the subject of esophageal hiatus hernia, for I have been interested in this condition for a number of years. I wish to compliment Dr. Humphreys and Mr. Collis upon their excellent presentations. Dr. Humphreys' analysis of surgical results of esophageal hiatus hernia of the diaphragm was very concise and comprehensive, and I compliment him upon the very good results obtained and the operative procedure he has utilized. I have not had the experience with the type of short esophagus, without associated esophagitis, that Mr. Collis has had. His series of 9 cases of this type is unusual. These cases present a different surgical problem than do other types of short esophagus, which he has solved by utilizing a relatively simple type of operative procedure upon the herniated portion of the stomach. I have not done an operative procedure of this type in these cases, but it seems to be a very practical procedure for this select group of cases. I compliment Mr. Collis upon his operative procedure, and the results he has obtained from it. I am greatly pleased that there is an increased interest in the subject of hiatal hernia in recent years. Thirty years ago it was difficult to stimulate an interest in this condition, because it was thought to be very rare. The condition is now known to be relatively common, and presents many interesting problems in diagnosis and treatment.

Esophageal hiatus hernia is primarily a mechanical condition and not a disease; however, it is not infrequently associated with complications such as esophagitis and gastric erosion and ulceration. I believe that the most important clinical consideration of hiatal hernias is the frequency with which the symptoms produced by this type of diaphragmatic hernia simulate the symptoms of other organic disease of the upper abdomen or lower thorax. This is also of surgical importance, for the symptoms so closely simulate those of other organic disease that surgical measures have been instituted for conditions other than the hernia without relief of symptoms. From a surgical standpoint, many different operative procedures have been advocated and are performed for hiatal hernias and the complications resulting from the herniation. This indicates that there is no one operation that is entirely satisfactory in all cases. There is not only difference of opinion as to methods of repair of the hernia, but also as to the method of approach. Some surgeons prefer the thoracic approach and some an abdominal approach. I prefer the abdominal approach because it has, I believe, special advantages for the repair of this type of diaphragmatic hernia; however, I believe this is the surgeon's choice, and he should select that type of approach which will enable him to repair most adequately the hernia, as well as correct any complications or disease that may be associated with the hernia.

Esophagitis is one of the most serious complications that occur in hiatal hernias; however, fortunately this complication does not occur in all cases. In the 489 cases of different types of hiatal hernia in which I have operated, esophagitis was present in varying degrees in 17 per cent of the cases. When esophagitis progresses to stricture formation, the treatment is directed at correction of the esophagitis, and usually requires an extensive operative procedure with considerable risk, and the result is not always satisfactory. I believe that the most important consideration of esophagitis is in its prevention rather than correction of the condition after serious structural changes have occurred in the esophagus.

This brings up what I believe is one of the most important surgical considerations of hiatal hernias at this time, which is the selection of cases for surgical treatment. At the clinic we have operated in about 18 per cent of the cases in which a diagnosis was made; this 18 per cent consists of patients in whom complications developed and those in whom symptoms were not relieved by conservative measures. I have considered for some years the advisability of operating upon a greater number of these hernias, even though they were causing only mild or no symptoms at the time they were found, so as to protect the patient from the possibility of subsequently developing complications from the herniation, especially esophagitis. I have not felt justified in this "complication-preventive" type of surgery, but I believe it should be considered if and when an operative procedure is developed for *hiatal hernia* which, when it is utilized, will enable the surgeon to assure the patient that there is less risk associated with the operative procedure than there is of the subsequent development of serious complications from the herniation, as well as little or no risk of a recurrence of the hernia. Again, I compliment the essayists upon their presentation.

DR. THOMAS H. BURFORD, St. Louis, Mo.—Like Dr. Sweet I am sorry that I did not have the brilliance or the imagination to think up this simple solution which Mr. Collis has presented. I am particularly grateful to Mr. Collis for telling us that he sees the same types of hiatal herniae in the British Isles that we see in the Midwest. Listening to my American colleagues, I had begun to fear that the fixed, *irreducible,* short esophageal type of hernia was seen only in St. Louis. To me the problem of the hiatal hernia comes down essentially to whether one is dealing with a sliding or reducible hernia, or with a fixed, so-called short esophageal type. With the former type there is, in my opinion, no real problem. In our experience, reduction and repair from above have been simple, safe, and permanent.

In the latter type, all efforts at reduction have failed and the so-called advancing operation where the esophagus is brought to the dome of the diaphragm has resulted in prompt return of reflux and progression of esophagitis in almost 100 per cent of cases. Because of this we sought to decrease reflux by balancing the resistance or the gradient at the pylorus with that at the incompetent cardia. To do this we chose the Finney type of pyloroplasty. These results were presented to the American Surgical Association last year, and were excellent. Since then more cases have been added and more time has elapsed. All patients have shown marked improvement with lack of symptoms and clearing of the esophagitis. I agree with Mr. Collis about the great importance of the angle of entrance or angle of implantation in the problem of reflux, and his solution appeals to me.

MR. R. S. PILCHER, London, England.—To me the problem in this disease is to know when the symptoms are due to hiatus, and when to operate. The syndrome which we recognize as characteristic of hiatus hernia was described many years ago by a pioneer of gastroenterology, Dr. Charles Bolton, and he was mainly concerned with the symptoms due to peptic ulcer. He recognized that in some cases what he called the esophageal syndrome was due to peptic ulcer, but he also noted that in many patients with this syndrome no ulcer was found. Having no knowledge of hiatus hernia he was led to dismiss these as hysteria, or psychoneurotic, and there is no doubt, I think, that, in many cases of hiatus hernia, symptoms have a psychoneurotic basis. There are a number of observations on the natural history of the disease which are, I think, interesting, because they bear on the statements made this morning. One of these is the spontaneous recovery of infants, to which Mr. Collis has already referred. Nevertheless, we sometimes see in infants a very rapid progression of esophagitis going on to severe stricture, and it is important to know which of these cases should be treated surgically. Children have something in common with very old people who get this trouble, and that is posture. It may be that the child's recovery is associated with the assumption of the upright posture, while the disease occurring in the elderly patient is sometimes due to resumption of the supine posture. We see patients who are being rehabilitated by orthopedic surgeons, flat on their back with traction on their legs, and in a few days they have esophagitis. There is not doubt that reflux can occur simply as a result of posture.

Another interesting finding in children is that those who have the large sliding hiatus hernia which usually occurs on the right with the fundus well above the cardia do not

have esophagitis. For that reason we thought that the solution of many of these problems was the restoration of angulation between the esophagus and the stomach. There are other interesting things about children; one is that they will be going along perfectly well and will suddenly get complete dysphagia and require urgent readmission. There are three things that will make a child do that: one is acute respiratory infection; another is bolus obstruction; and the third is frustration. A child who is going along quite well may have a sudden relapse from any of these causes.

That brings me to a similar observation in adults. Sometimes an adult with a hiatus hernia gets symptoms only when he is disturbed or upset, and we have had patients who were perfectly all right until they had an important business engagement or some public speaking to do, and then the cardiac played up.

Another thing in children is the significant association between the symptoms we would attribute to hiatus hernia, and hypertrophic pyloric stenosis, and it is always important, I think, when we find hiatus hernia or reflux to make sure that the patient does not have pyloric stenosis. Sometimes what we might call the hiatus hernia or esophageal syndrome is shown by x-ray to be due to pyloric stenosis. Hiatus hernia is not a new disease, and we know that many cases are picked up in which the patients have no symptoms referable to that disease. It is the critical part of the problem to know when the symptoms can be attributed to hiatus hernia and when to other conditions.

DR. GEORGE H. HUMPHREYS, New York, N. Y. (Closing).—I should like to echo Dr. Harrington's statement that it is good to have so much discussion on this subject, which is a sort of poor relation to chest surgery in general. I am gratified by Dr. Sweet's comment, since he has had so much more experience than I, and by Dr. Lam's and Dr. Harrington's also. How to make the diagnosis of recurrence? This is a question I cannot answer any better than he can—I have to take what the x-ray man gives me as a recurrence. I take it not always willingly, especially when the patient has no symptoms. However, it is not without some validity because it does represent, I am sure, at least some reflux. Whether the actual object the x-ray man sees is an epiphrenic ampulla or a true recurrence, it represents, nevertheless, to a certain extent failure of the operation because there is some gastric reflux.

On the question of moving the esophagus to the dome of the diaphragm, I agree with the other discussants that this is an operation which should be abandoned. It is a useful maneuver if it can be done to reduce the hernia, but it usually results in continued reflux and sometimes in stricture of the esophagus if the diaphragm is closed too tightly. Certainly Mr. Collis' operation is a far better one if the esophagus is permanently shortened. I think that the persistent esophagitis symptoms, however, are not due necessarily to persistent reflux, but after a time the esophagus goes through inflammatory changes which are irreparable, and even though the angle is restored and there is no further reflux, the symptoms continue because of the disease in the esophagus itself. I see no relief for this except resection of the esophagus and possibly the very ingenious operation of using the ileocolic valve might come in here.

It is certainly true that pyloroplasty may be a useful adjunct in these cases and Dr. Burford has made a real contribution here.

Mr. Pilcher raises the most important question of all—when are the symptoms due to hernia and when should we operate? The only way I can answer him is to ask, when are the symptoms of inguinal hernia due to the hernia, and when should we operate?

MR. J. LEIGH COLLIS, Birmingham, England (Closing).—I should like to thank you for the kind things that were said about my paper.

Whatever type of operation is used for any hiatus hernia, it is essential that gastric reflux must be controlled. Dr. Grimes has put forward an ingenious plan which would fulfil this requirement, but it would seem likely to suffer from being too complicated and too large an operation. Dr. Kay's operation is well designed for the control of reflux and has the great virtue of simplicity, but it does seem esthetically wrong to leave so much stomach in the chest.

Once again I should like to thank you for the kind reception you have given my paper and for the great honor you have done me in letting me take part in this meeting.

# TRAUMATIC RUPTURE OF THE DIAPHRAGM

## Clinical Manifestations and Surgical Treatment

Gerard Desforges, M.D.* (by invitation), John W. Strieder, M.D.,** Joseph P. Lynch, M.D.,*** and Irving M. Madoff, M.D.****
Boston, Mass.

DIAPHRAGMATIC rupture may be due to many types of trauma such as gunshot and stab wounds, as well as blunt injury. It may also be secondary to prolonged subphrenic septic processes. This presentation is concerned with only that type of diaphragmatic rupture which is secondary to impact injury.

The increase in automobile travel has brought with it the all too familiar problem of multiple serious injuries. Not the least of these is blunt thoracoabdominal trauma which, in addition to visceral injuries, may result in traumatic rupture of the diaphragm. The automotive crash injury research project at Cornell University Medical School reports that of a group of 1,678 injured occupants of 1,000 automobile crashes, 65 per cent sustained injuries of two or more body areas.[11] It is among these patients with multiple injuries that ruptured diaphragm is most prone to occur. It is also under such circumstances that the evidence of diaphragmatic rupture is obscured, or it may be part of an aggregate of symptoms difficult of interpretation.

The authors have had occasion to treat 16 cases of rupture of the diaphragm seen both early and late in relation to an episode of trauma. Certain clinical features, diagnostic characteristics, and operative findings have been of sufficient interest to warrant analysis of our data.

### CASE REPORTS

Case 1.—C. L., a 6-year-old boy, was run over by an ice truck, the two rear wheels passing over his abdomen. He was immediately taken to the Boston City Hospital where shock and marked shortness of breath were noted. Roentgenograms revealed a fractured pelvis and irregularity of the left diaphragm. After transfusion and gastric aspiration, the patient was much more comfortable, but serial films of the chest revealed a progressively enlarging gastric shadow in the left hemithorax. Nasogastric suction prevented severe gastric dilatation and the patient's general status improved. On the eleventh day, transthoracic repair of the ruptured left diaphragm was carried out. A large rent in the diaphragmatic dome was found. The eviscerated organs were stomach, spleen, and portions of colon and small bowel. Repair was done with imbricating silk sutures. Recovery was uneventful.

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Read at the Thirty-seventh Annual Meeting of The American Association for Thoracic Surgery at Chicago, Ill., May 4-7, 1957.

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****Instructor in Surgery, Boston University School of Medicine.

CASE 2.—A. C., a 29-year-old man, was struck by a car. He sustained multiple fractured ribs on the left and generalized abrasions and contusions. On admission to the Massachusetts Memorial Hospitals, the most marked symptom was shortness of breath. Chest roentgenograms revealed what was thought to be bowel shadows in the left hemithorax. Operation through the left chest was done on the second hospital day. The diaphragm appeared to have been lacerated in its lateral portion by an inward projecting fractured rib end. Through a relatively small rent, about one inch long, a portion of omentum and splenic flexure of colon had herniated. The omentum was hemorrhagic and so was resected. Through a counter incision, the colon was drawn back into the abdomen and both diaphragmatic apertures closed. Convalescence was smooth and the patient was discharged from the hospital on the tenth postoperative day.

CASE 3.—J. H., a 40-year-old man, was a passenger in an automobile which was struck by another car. On admission to the Winchester Hospital, the most striking findings were dyspnea, cyanosis, and shock. Roentgenograms revealed a fractured clavicle, fractured pelvis, and a "very high left diaphragm." Films with Levin tube in place revealed the latter to be in an intrathoracic stomach. Surgery was done on the second hospital day at which time a 5-inch rent in the dome of the diaphragm was repaired transthoracically. A major portion of the stomach had herniated into the chest. The patient convalesced uneventfully.

CASE 4.—P. W., a 64-year-old man, fell on the street after a drinking bout and was brought into the Carney Hospital nauseated and dyspneic. He complained also of pain in the left chest radiating to the back. Peristaltic sounds could be heard on auscultation of the left chest and the trachea was deviated to the right. Both large and small bowel were demonstrated to be within the thoracic cavity by barium enema and upper gastrointestinal series. The patient refused surgery and was lost to follow-up.

CASE 5.—E. P., a 36-year-old man, fell 30 feet from a scaffolding. At the time of admission to the Quincy City Hospital he was experiencing severe respiratory difficulty and was semiconscious. Roentgenograms revealed a fractured left femur and a fractured skull. A nasogastric tube was introduced to relieve a rapidly distending intrathoracic stomach, but it could not be made to function properly. Because of the patient's poor condition, a laparotomy was done in an attempt to repair the ruptured diaphragm. A transverse rent was found and stomach, spleen, large bowel and small bowel were reduced. During the repair, cardiac arrest occurred and the patient died in spite of resuscitative measures.

CASE 6.—O. B., a 30-year-old sailor, was injured as a passenger in an automobile which went out of control while traveling at high speed. On admission to the Norwood Hospital, he had severe respiratory distress, evidence of head injury, and a fracture of the left femur. Chest films revealed "contusion" of right lung and a high diaphragm on the left. Repeat films revealed an enlarging gastric gas shadow. On the second hospital day, during the transthoracic repair of a ruptured left diaphragm with a major portion of the stomach in the chest, cardiac arrest occurred. Massage restored a normal rhythm, but postoperatively, the patient did poorly and died on the third day following surgery.

CASE 7.—F. C., a 51-year-old woman, was a passenger when the vehicle in which she was riding was struck by another car. She was brought to the Boston City Hospital in severe respiratory distress. Auscultation revealed bowel sounds in the left chest and roentgenograms showed mediastinal shift to the right, with a large gastric shadow in the left hemithorax. The diagnosis of ruptured diaphragm was made and operation performed on the first hospital day.

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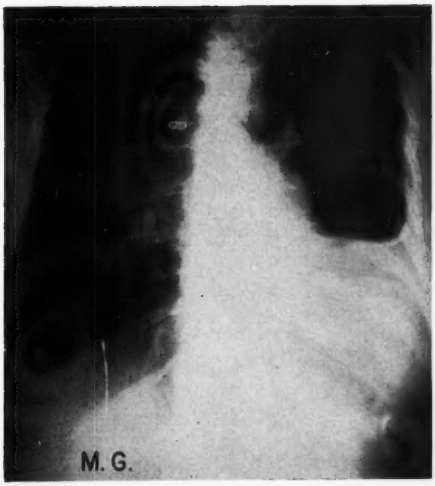
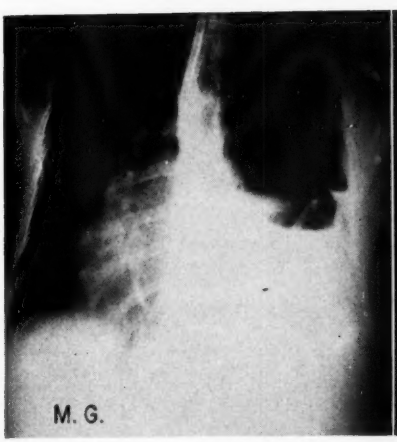
Fig. 1.—Patient M. G. (Case 8). *A*, (Dec. 1, 1953) Film of chest taken 5 days after trauma reveals large gas shadow in left lower chest.

*B* and *C*, (Dec. 7, 1953) Suspicion of ruptured diaphragm confirmed by use of Levin tube and barium swallow.

*D*, (Dec. 21, 1953) Postoperative chest roentgenogram.

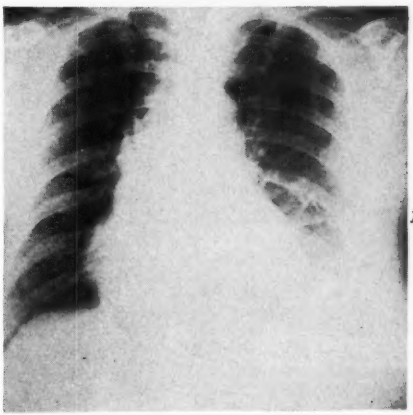



A.




B.

C.

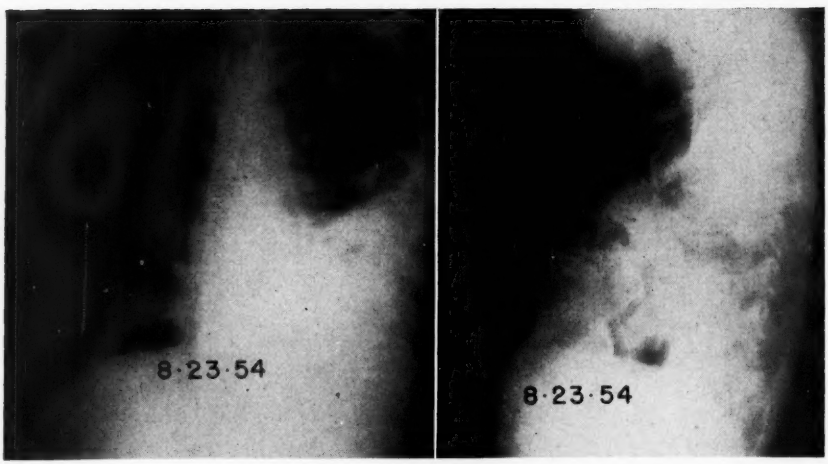


D.

Fig. 1.—*(For legend, see opposite page.)*

A nasogastric tube which had been placed previously was never made to function properly. At the time of operation, decompression of the stomach was done by introduction of a needle into the markedly distended viscus. It was found that the stomach had eviscerated through a 4-inch rent from the mid-pericardial level laterally through the dome of the diaphragm. In addition to the stomach, omentum and large bowel were also found in the chest. These were replaced into the abdomen and laceration repaired. The patient did poorly following surgery and died 24 hours later in respiratory distress secondary to continual accumulation of secretions.

*A.*



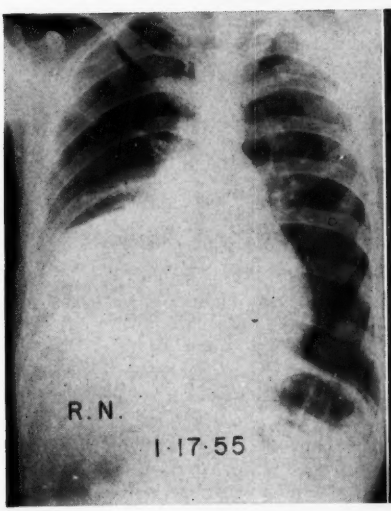

*B.* *C.*

Fig. 2.—Patient P. B. (Case 10). *A*, (Aug. 15, 1954) Flat film of abdomen taken on admission reveals large bowel pattern and gaseous distention.

*B* and *C*, (Aug. 23, 1954) Posteroanterior and lateral views of barium studies showing splenic flexure extending into chest.

Fig. 3.—Patient R. N. (Case 12). *A*, (Dec. 18, 1954) Chest roentgenogram on admission showing homogeneous density in right hemithorax plus mediastinal shift. *B* and *C*, (Jan. 17, 1955) Posteroanterior and lateral roentgenograms of chest after thoracentesis. Diaphragmatic eventration was suggested but note lack of sharp costophrenic angle. *D*, (Feb. 1, 1955) Postoperative chest roentgenogram.

A.




B.

C.

D.

Fig. 3.—*(For legend, see opposite page.)*

CASE 8.—M. G., a 64-year-old woman, was thrown from a car in collision with another automobile. On admission to the Lawrence Memorial Hospital, she was markedly short of breath and had severe pain in her lower abdomen. Under observation, a fractured pelvis was discovered by roentgenograms, and serial chest films revealed a progessively enlarging gastric shadow in the left hemithorax. Films, with Levin tube in place (Fig. 1) and after barium swallow, confirmed the suspicion of a ruptured diaphragm with an intrathoracic stomach. At operation through the left chest on the eleventh hospital day, a T-shaped laceration of the diaphragm was found with the transverse limb along the pericardial attachment and the perpendicular limb extending laterally into the dome of the diaphragm. Transthoracic repair was carried out easily and convalescence was uneventful.

*A.*

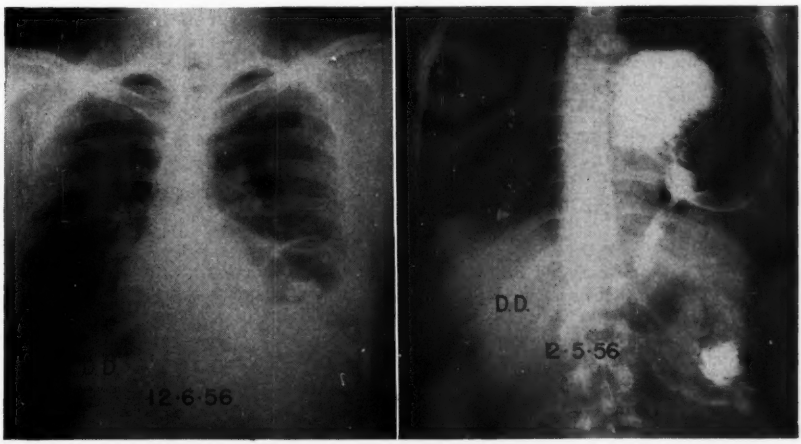
Fig. 4.—Patient D. D. (Case 13). *A*, (Feb. 16, 1952) Chest roentgenogram taken 22 years after original trauma. Patient asymptomatic. Diaphragmatic eventration was suggested but costophrenic angle not sharp.

CASE 9.—J. B., a 36-year-old man, was a passenger in an automobile struck by another and was brought into the Carney Hospital with marked difficulty in breathing. Films taken on admission revealed a question of a large gas bubble in the left lower chest and a fractured pelvis. Serial films showed that the left lower chest gas bubble was gradually increasing in size and a suspicion of a ruptured diaphragm with intrathoracic stomach was confirmed by repeat roentgenograms with a Levin tube in place. Exploration through the left pleura was performed on the fourth day and a large portion of the stomach was found within the chest. Reduction was effected and the diaphragmatic rent was repaired. The patient recovered uneventfully.

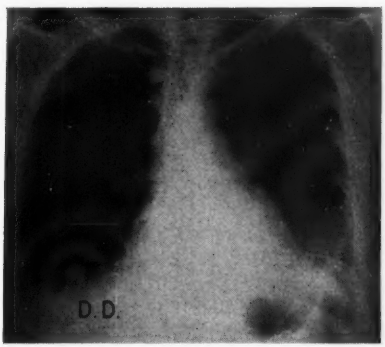
CASE 10.—P. B., a 36-year-old man, was brought into St. Elizabeth's Hospital with abdominal distention and signs of large bowel obstruction. This was confirmed by flat films of the abdomen and it was felt that the patient, in spite of his age, probably had a malignant splenic flexure tumor. Emergency cecostomy was done on admission to the hospital. When barium enema studies were carried out (Fig. 2), it was evident that the splenic flexure extended into the chest. During this study, it was also evident that this man had fractures of his lower ribs. At this time, thoracic surgical consultation was requested. The patient stated that he had been in an auto accident 3 months previously and he knew that he had incurred fractured ribs at the time. Transthoracic repair of the diaphragm was carried out uneventfully following restoration of colon to the abdomen. The cecostomy was allowed to close and the patient was discharged on the twentieth hospital day.

CASE 11.—A. M., a 57-year-old man, fell 30 feet down an elevator shaft and was brought into the Boston City Hospital in shock with severe pain in his right arm and over

his pelvis. X-rays revealed fractured pelvis, fractured lumbar vertebrae, a fractured right humerus, and fractured ribs on the left. Roentgenograms of the chest revealed an elevated left diaphragm and this "elevation of diaphragm" grew progressively higher in the following several days. On the fifth day after admission, thoracic surgical consultation was sought. The diagnosis of a ruptured left diaphragm was made and repair with restoration of a large portion of herniated stomach was carried out transthoracically. The patient tolerated the procedure well and convalescence was uneventful.




*B.* *C.*




*D.*

Fig. 4. (Cont'd).—*B*, (Dec. 6, 1956) Barium enema study showing colon extending into chest.

*C*, (Dec. 5, 1956) Upper gastrointestinal series reveals almost all of stomach in left hemithorax. Note, also, that while swallowing barium, patient began to markedly distend a portion of stomach into which barium did not enter.

*D*, (Jan. 18, 1957) Postoperative roentgenogram of chest.

CASE 12.—R. N., a 20-year-old man, was standing behind one automobile when he was struck by another traveling at high speed. He sustained fractures of both arms, both legs, and of the pelvis. On admission to the Melrose Hospital, chest roentgenograms showed an extensive homogeneous density on the right (Fig. 3). Thoracentesis done at the time

of admission produced 2 L. of bloody fluid. Following the thoracentesis, the patient's dyspnea was markedly improved, but serial chest films revealed a persistent density in the lower right hemithorax. Thirty days following the accident, right thoracotomy was performed and an extensive laceration of the diaphragm was found. This extended from the paravertebral level, posteriorly, to the ribs, anteriorly. Most of the liver had herniated into the right chest. The liver was easily replaced. The rent was repaired and the postoperative convalescence was uneventful.

CASE 13.—D. D., a 39-year-old man, had been run over by an automobile 26 years previously. Up to 2 years prior to consultation, the patient had felt fairly well, but during the previous 2 years, he had been troubled by upper gastrointestinal symptoms. At the time of consultation, the patient could not finish a meal without stopping to bend over

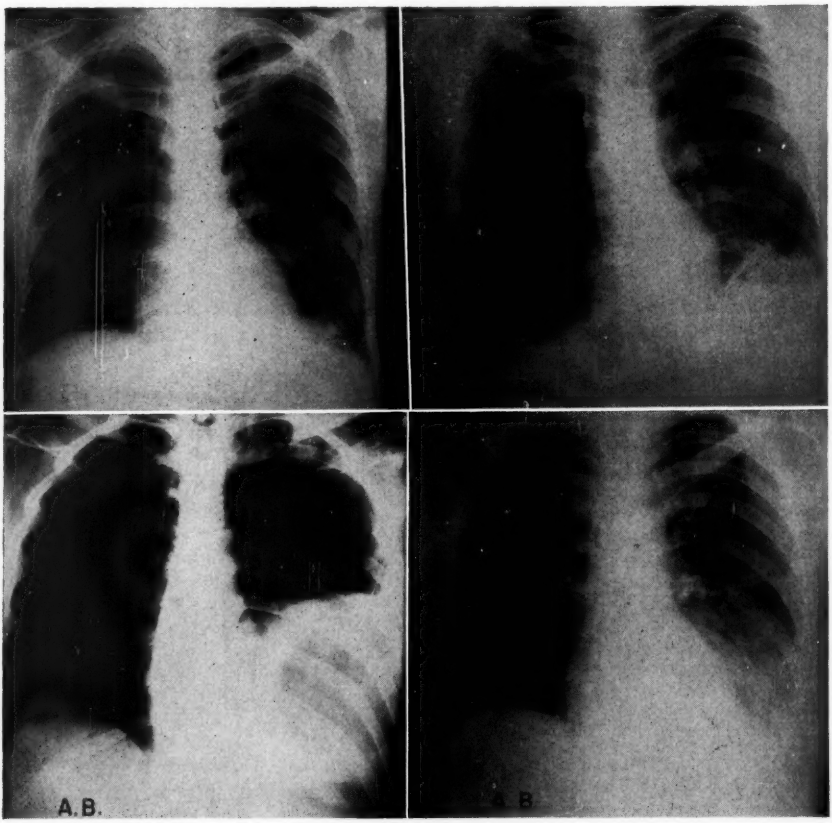



Fig. 5.—Patient A. B. (Case 15). *A*, (April 10, 1950) Admission roentgenogram of chest at the time of trauma showing only irregularity of left diaphragm.

*B*, (Nov. 12, 1956) Six years later chest roentgenogram reveals "elevation" of left diaphragm.

*C*, (Nov. 21, 1956) At this point, signs of high intestinal obstruction are present and chest roentgenogram reveals large portion of distended stomach within chest.

*D*, (Nov. 27, 1956) Postoperative chest roentgenogram.

a chair to raise gas by belching. Because of these worsening symptoms, the patient was studied by upper gastrointestinal series and barium enema. It was evident from these studies, considering the history of trauma, that this man had a ruptured diaphragm (Fig. 4) with stomach and colon in the left hemithorax. Surgical intervention and repair was recommended. At operation, at the Charles Choate Memorial Hospital, colon and omentum were found in the left pleura with marked compression of the left lung. The diaphragmatic rent was repaired. Between the aorta and the esophageal hiatal ring, there was a defect which could not be closed. To close this defect, a pedicled pericardial flap was brought down and sutured into position (see Fig. 11, *D*). This made a most satisfactory closure. The patient recovered uneventfully and was discharged from the hospital on the fifteenth hospital day.

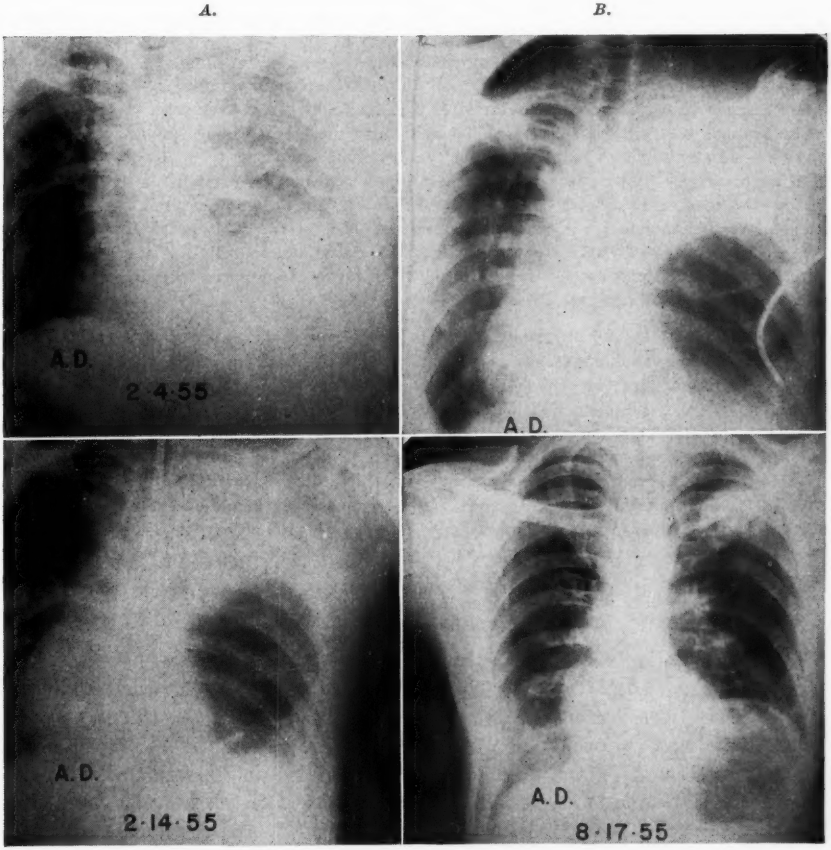



Fig. 6.—Patient A. D. (Case 16). *A*, (Feb. 4, 1955) Chest roentgenogram on admission showing "elevation of left diaphragm."

*B*, (Feb. 12, 1955) As gaseous shadow, interpreted as pneumothorax, enlarged, multiple thoracenteses were done and finally closed thoracotomy was carried out. Roentgenogram now reveals large intrathoracic gastric gas shadow with lung compression and mediastinal shift. Closed thoracotomy catheter in place.

*C*, (Feb. 14, 1955) Suspicion of diagnosis of ruptured diaphragm confirmed by chest roentgenogram with decompressing Levin tube in place.

*D*, (Aug. 17, 1955) Postoperative chest roentgenogram.

CASE 14.—J. H., a 23-year-old white woman, was seen because of an abnormal chest film taken during a routine survey. In her previous history, it was known she had been in an automobile accident 4 years previously, at which time she was hospitalized for extensive soft tissue injuries, plus a fractured pelvis, and a fractured left hip. Confirmation of the suspicion of a ruptured left diaphragm was made by both upper gastrointestinal series and barium enema. At left thoracotomy at the New England Baptist Hospital, a large laceration was found through the dome of the left diaphragm. The herniated left lobe of liver, colon, small bowel, stomach, omentum and spleen were restored to the abdomen and the rent repaired by imbricating silk sutures. Convalescence was uneventful.

CASE 15.—A. B., a 36-year-old man, was seen because of symptoms of upper gastrointestinal distress and vomiting. There was also pain in the left shoulder after eating a large meal. His history revealed that he had been in an automobile accident 6 years previously at which time he suffered cerebral concussion, fracture of the left tibia, and contusion of the chest. Chest roentgenograms at the time of the original injury revealed no abnormality, except a slight irregularity of the left diaphragm (Fig. 5). Because of persistent upper gastrointestinal symptoms for 2 years following his accident, the patient had had an upper gastrointestinal series done with no abnormality being found. However, at the time of admission, 6 years following the accident, symptoms of acute gastric obstruction were severe. Upper gastrointestinal series now revealed a large portion of the stomach within the chest with a "closed loop" obstruction. A diagnosis of ruptured diaphragm was made. Transthoracic repair was carried out at the Lawrence General Hospital as an emergency because of clinical deterioration. Beginning strangulation of the stomach was found. This herniated viscus was reduced. The large diaphragmatic rent was repaired. Convalescence was uneventful.

CASE 16.—A. D., a 59-year-old man, was the driver of an automobile which struck a tree. At the time of admission to the Anna Jacques Hospital, he was in shock, was found to have sustained fractures of both lower legs, pelvis, and both forearms. Plain serial roentgenograms of the chest (Fig. 6) revealed a progressively enlarging gas shadow in the left lower chest. This had been tapped repeatedly on the suspicion that it was a pneumothorax. The patient had also been subjected to a closed thoracotomy for a decompression of this "pneumothorax." When these methods failed, thoracic surgical consultation was sought. A diagnosis of ruptured diaphragm was immediately made and repair carried out. Early convalescence was somewhat stormy because of difficulty with secretions. These were handled satisfactorily by repeated endotracheal catheter suction and after the first week convalescence proceeded uneventfully.

## ANALYSIS OF DATA

Sixteen patients whose ages range from 6 to 64 years were seen. There were 13 males and 3 females. The injuries leading to diaphragmatic rupture in this series were automobile accidents or falls. Nine of the 16 cases of diaphragmatic rupture occurred in people who were either drivers or passengers in motor vehicles. Four patients were struck by a motor vehicle and 3 sustained their trauma as the result of a fall (Table I). Of these 16 cases, only one did not have associated serious skeletal injury. Eight patients sustained a fractured pelvis and in 8 patients there were also fractured ribs. Seven of the 16 sustained fractures of the long bones or the spine. Three patients sustained concomitant head injuries. Some of the patients had combinations of all these categories of associated trauma (Table II). The most prominent symptom of these patients with traumatically ruptured diaphragms was shortness of breath and this occurred in 11 of the 16 patients (Table III). Evidence of cardiopulmonary insufficiency was most striking in the early posttraumatic

period. Analysis of other signs and symptoms such as cyanosis, grunting respiration, and rapid pulse led to the belief that these were manifestations of severe generalized trauma.

TABLE I. ETIOLOGIC FACTORS

| | |
|---|---|
| Struck by vehicle | 4 |
| Passenger or driver | 9 |
| Fall | 3 |
| Total | 16 |

TABLE II. ASSOCIATED TRAUMA

| | |
|---|---|
| Fractured pelvis | 8 |
| Fractured ribs | 8 |
| Fractures of long bones and/or spine | 7 |
| Head injury | 3 |

TABLE III. SYMPTOMS WHEN PATIENTS SEEN

| | |
|---|---|
| Dyspnea | |
| all left sided ruptures, seen early (less than 2 weeks) | 11 |
| Gastrointestinal | |
| all left sided, seen late | |
| a. vomiting | 2 |
| b. obstipation | 1 |
| None | |
| a. late left (4 years) | 1 |
| b. early right (2 weeks) | 1 |

Two patients had no symptoms referable to their ruptured diaphragm and that abnormality was suggested by plain films taken for other purposes. Three patients had gastrointestinal symptoms. Vomiting was present in 2 of these. One of these had left shoulder pain which was precipitated by eating a heavy meal and one patient developed acute large bowel obstruction. All of these patients with gastrointestinal symptoms were seen late in relation to their traumatic episode—3 months, 6 years, and 26 years, respectively. Two of these 3 had acute gastrointestinal obstructive symptoms requiring emergency surgery.

Most cases were seen within a few days of the accident. Eight patients were operated upon within the first 5 days of injury, 4 of these having been seen on the second day. Four others were operated upon within 30 days of the trauma. The remaining 4 patients were operated upon in 3 months, 4 years, and 26 years, following their injury. At operation, a variety of abdominal viscera were found within the thorax. The one right-sided rupture revealed only a major portion of liver within the right chest, but on the left, the abdominal thoracic contents varied from stomach alone, to stomach, large and small bowel, spleen and omentum and liver (Table IV). There was no way of telling preoperatively what the thoracic contents, other than the stomach and colon, would be.

In this series of cases of ruptured diaphragm, the most important diagnostic aids were history and serial plain roentgenograms of the chest. These two measures led to the diagnosis in 13 of the 16 cases. Chest roentgenograms

made with a Levin tube introduced into the stomach helped to make the diagnosis in one patient and barium studies were required in 2 patients (Table V).

TABLE IV. ORGAN EVISCERATED AND SIDE OF RUPTURE

| | | |
|---|---|---|
| Through Right Diaphragm | | 1 |
| Liver | 1 | |
| Through Left Diaphragm | | 15 |
| Stomach | 3 | |
| Stomach and omentum | 7 | |
| Colon and omentum | 1 | |
| Stomach, colon, small bowel, spleen | 1 | |
| Stomach, colon, small bowel, omentum | 2 | |
| Stomach, colon, small bowel, spleen, omentum, liver | 1 | |

TABLE V. DIAGNOSTIC METHODS

| | |
|---|---|
| History of trauma | 16 |
| Chest roentgenograms, serial | 13 |
| Chest roentgenogram plus Levin tube | 1 |
| Chest roentgenogram plus barium studies | 2 |

There were 3 deaths in this group of 16 cases. Of the 3 who died, 2 had very serious head injuries. The third death was in a 51-year-old woman, who had great difficulty with tracheobronchial secretions, both before and after operation, and eventually died of respiratory failure.

## DISCUSSION

It is apparent that all of these patients suffered from some form of severe impact trauma. Almost all of the patients who had diaphragmatic rupture secondary to blunt trauma were victims of automobile mishaps or falls. Although the decelerative type of injury has been described as being a common cause of this trauma,[5, 7, 10, 12] it seems from this series that any type of blunt thoracoabdominal trauma may give rise to diaphragmatic rupture. It would appear that the relatively weak diaphragmatic structure yields between the solid viscera of the abdomen and the pliant thoracic structures as the result of sudden force applied to either the thoracic or abdominal wall (Fig. 7). The direction of the trauma per se does not seem important in the disruptive mechanism. It appears, however, that force directed to the lumbar region is infrequently associated with rupture of the diaphragm.[6] Forces applied to the anterior abdomen and the flanks are transmitted according to Archimedes principle, equally in all directions, and the diaphragm, offering the least resistance, fails by disruption. The liver may act as a buffer protecting the right hemidiaphragm which is rarely ruptured.[1, 13, 15-21] In all series reviewed and in our own, right-sided diaphragmatic rupture occurred only about one twentieth as frequently as rupture on the left.

When the forces are applied to the thoracic cage with consequent deformity of the thoracic contour, the diaphragm again appears to be a structure of relative weakness and tears can be produced by force applied in the anteroposterior or lateral direction. A crude analogy to this particular mechanism

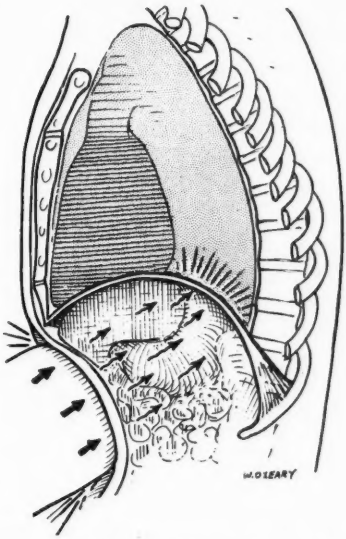



Fig. 7.—Schematic representation showing abdominal trauma with theoretical mode of production of diaphragmatic disruption.

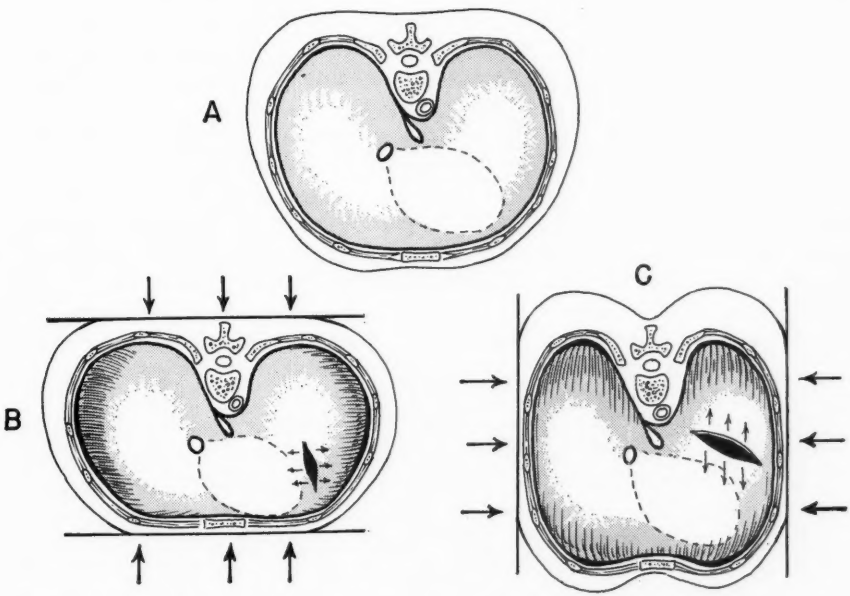



Fig. 8.—*A*, Normal diaphragmatic contours. *B*, Diaphragm exposed to forces in anteroposterior directions. *C*, Diaphragm exposed to forces in lateral directions.

is suggested by a wet piece of paper stretched over a hoop.[15] With distortion, this friable drumhead will rupture (Fig. 8). It seems logical to assume that this type of injury is the one that can more easily produce the right-sided rupture with herniation of the liver into the right pleural cavity. Case 12, R. N., is an example of right-sided rupture secondary to crushing between two automobiles (see Fig. 3).

The clinical features of the ruptured diaphragm are frequently obscured by other associated serious injuries.[2, 3, 4, 8, 9] The suspicion of the existence of a ruptured diaphragm may readily lead to the explanation of confusing findings and the correct therapy may be instituted. In this connection, 4 of our patients had multiple thoracenteses performed under the misapprehension that pneumothorax was present when in reality the space being tapped was a distended stomach herniated into the thorax. Indeed, Case 16, A. D. (see Fig. 6), was treated with closed thoracotomy prior to consultation. This therapeutic misadventure has been reported by others.[14]

The clinical picture of diaphragmatic rupture may be conveniently divided into three phases: (1) an acute, or immediate phase, (2) a chronic phase, and (3) an intermediate or dramatic phase.[3, 4] In the acute phase, the signs and symptoms of ruptured diaphragm depend at least to some extent on the size of the tear and the quantity and character of the displaced viscera if any. Obviously, a small tear will produce a few or no signs or symptoms of severity adequate to stimulate the physician to entertain the diagnosis, particularly when there are other serious concomitant injuries. However, even in this situation, a roentgenogram of the chest may demonstrate a slight elevation of the diaphragm or a shadow suggesting a small pleural effusion difficult of explanation on the basis of history and physical findings. Knowledge of the history of flank and/or abdominal trauma, together with such a chest film, may lead to the diagnosis of diaphragmatic rupture.

Roentgen examination is the single most valuable tool in confirming the suspicion of diaphragmatic rupture. A posteroanterior roentgenogram of the chest, preferably in the upright position, combined with a history of trauma should, in most instances, suggest this possibility. Obliteration of the diaphragmatic angle, deformity of the diaphragmatic contour, "atelectasis of the lower lobe," "pneumothorax in the lower chest," all suggest the diagnosis of ruptured diaphragm. The presence of fractured ribs is not a necessary part of the diagnostic picture. Having considered the possibility on the basis of the history, and roentgenograms of the chest, the diagnosis may be substantiated by several maneuvers. First, a simple technique of repeating chest roentgenograms after the introduction of a nasogastric tube may often indicate the intrathoracic portion of the stomach (see Fig. 1). Further use of contrast media, showing the abnormal position of the stomach and colon, will serve the same purpose (see Fig. 1). Occasionally, barium will not pass into the intrathoracic portion of the stomach because of obstruction at the site of tear secondary to torsion of the stomach (see Fig. 5). Therefore, it would seem that barium studies alone are not to be relied upon. When the

right diaphragm is involved, the above techniques may not be helpful. However, barium enema may show an elevated hepatic flexure. Although we have not yet had an opportunity to use the technique ourselves, we suggest the use of intravenous radiopaque cholangiographic dye to delineate the liver in its position with relation to the intrathoracic cavity. Fluoroscopy for paradoxical motion may be misleading, since, in one instance of right-sided rupture, it was found that the displaced liver moved with respiration during this type of examination (Case 12, R. N.).

In general, the suspicion of extensive diaphragmatic disruption, in the acute phase, is borne out by impressive symptoms such as dyspnea, cyanosis, and shock. Chest roentgenograms show abnormal intrathoracic shadows and in many cases mediastinal shift with compression of the ipsolateral lung (see Fig. 1). Early diagnosis is important since the injury may require early surgery.

The chronic phase of diaphragmatic rupture is exemplified by those patients who at some time in the past have had abdominal or thoracic trauma of relatively serious nature and who at a later date present symptoms suggestive of a wide variety of upper abdominal disorders. Case 13, D. D., suffered injury 26 years previously when struck by a truck. He remained well for approximately 24 years and then began to experience upper abdominal distress particularly on eating.

Diagnosis from history alone is in the nature of a philosophical discussion. However, with diagnostic adjuncts it should become apparent. These patients may complain of epigastric distress, gaseous eructation, postprandial discomfort, and obstipation. The sensation of shoulder discomfort when related to eating may point toward diaphragmatic involvement. Obviously such studies as barium swallow, upper gastrointestinal series, and barium enema will help delineate the pathology. Occasionally, ruptured diaphragm may be confused with diaphragmatic eventration. In this situation a roentgenogram may show a sharp costophrenic angle when there is a diaphragmatic eventration whereas, with rupture, the peripheral margin of the "diaphragm" is rarely sharp (see Figs. 3, *B*, and 4, *A*).

An intermediate or dramatic phase of diaphragmatic rupture announces itself with the signs and symptoms of high or low intestinal obstruction. Case 10, P. B., and Case 15, A. B., show these respective syndromes.

This symptomatology depends on what portion of the gastrointestinal tract has become incarcerated in the diaphragmatic rupture. In this phase, immediate operation is mandatory and frequently must be done under less than ideal circumstances. These patients are usually well for a longer or shorter period of time after the traumatic incident. The acute episode may be looked upon as something totally unrelated to the episode of trauma. Understandably, this lack of recognition of relationship will be inversely proportional to the length of time from the original accident. If the episode or type of trauma is borne in mind, what appears as a strange pathophysiologic picture immediately resolves itself into a tentative diagnosis readily

confirmable. The signs and symptoms of gastric or intestinal obstruction are nonspecific. However, major physical findings of significance may be dullness, tympany, or borborygmi in the involved hemithorax.

As in all disease entities, there is no substitute for a high index of intelligent suspicion. The use of radiologic adjuncts obviously must depend on the patient's clinical status. Immediately following trauma, nothing but the simplest of techniques may be undertaken. By the same token, patients with chronic intestinal symptoms, in whom such a diagnosis is being considered, must be submitted to all of the previously mentioned modalities of examination before dismissing the possibility of ruptured diaphragm. The establishment of a diagnosis in these patients will forestall the development of obstructive symptoms and the institution of surgery under less favorable conditions.

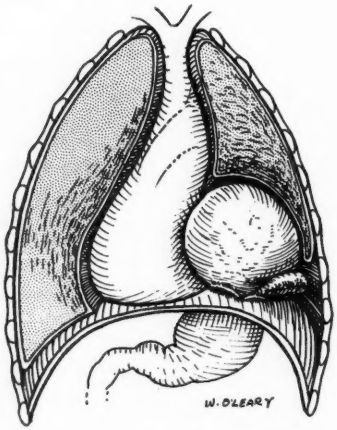



Fig. 9.

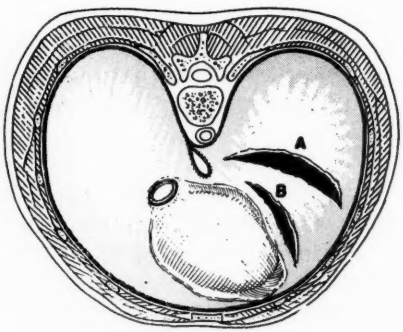



Fig. 10.

Fig. 9.—Diaphragmatic representation of compression of ipsilateral lung, mediastinal shift, and compression of contralateral lung, also by the herniated and distended viscera.

Fig. 10.—Common sites of diaphragmatic rupture—through the dome or in the parapericordial positions.

It is also apparent that after a period of several years have passed, the patient has usually lost his opportunity for financial redress for the portion of his injuries which was not recognized at his original hospitalization. This results in an unfair financial burden because subsequent hospitalization costs usually must be borne by the patient. Four of the patients reported in this group of cases fell into this category.

Once the diagnosis has been established in the acutely injured, management is basically a matter of timing. In the seriously ill patient, the victim of multiple injuries, it is often difficult to decide which single injury should receive priority. Certainly, it is a basic precept that treatment of pain, shock, and hemorrhage must take precedence. With due regard for the fundamentals of the care of trauma, the symptoms which lead to the need for early repair

of a ruptured diaphragm are dyspnea, cyanosis, and evidence of cardiac embarrassment due to mechanical displacement (Fig. 9). Unless the clinical picture produced by displaced viscera is such that the patient's life is in jeopardy, surgical intervention should be deferred until an early elective period. In this latter situation, the simple maneuver of gastric decompression may ameliorate the patient's symptoms so that he can be quite comfortable during a somewhat prolonged period of physiologic restoration.

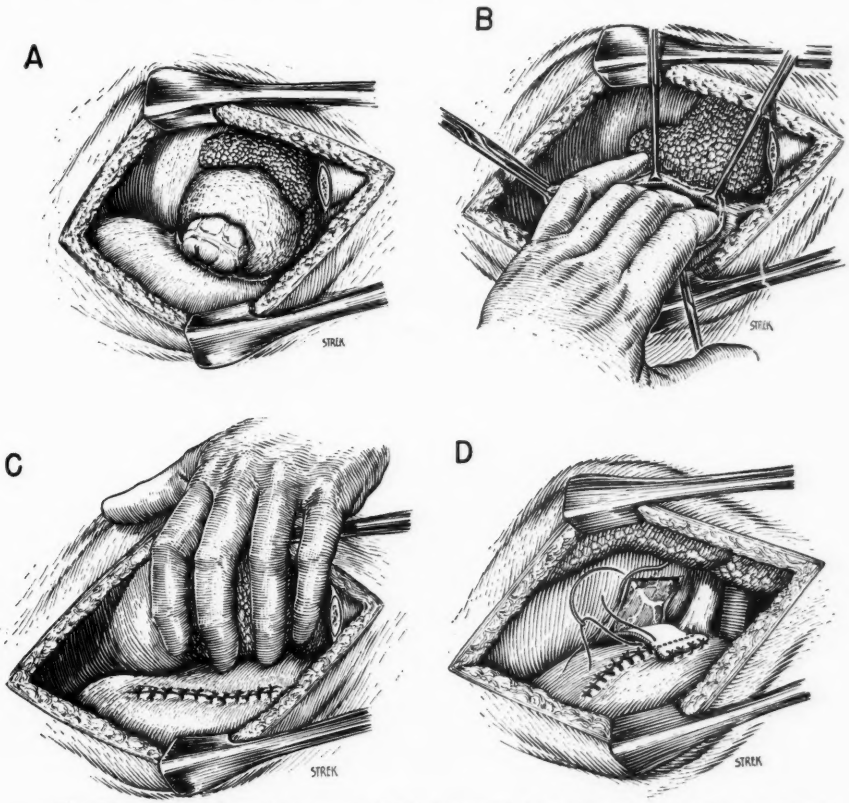



Fig. 11.—*A*, Transthoracic approach to diaphragmatic laceration shows excellent exposure that can be obtained.
*B*, Displaced viscera returned to abdominal cavity.
*C*, Closure of diaphragmatic rent with interrupted silk sutures.
*D*, Method of closing persisting diaphragmatic defects with pedicled pericardial graft.

When the decision to operate has been taken, thoracotomy, in our hands, has the virtues of simplicity and easy access to the lesion. Using the lateral decubitus position, the eighth rib may be resected or the pleural space entered through the eighth intercostal space, with or without division of ribs 8 and 9 posteriorly. The usual positions of laceration are parapericardial or through the diaphragmatic dome (Fig. 10). Occasionally, there may be an avulsion of

the diaphragm from its lateral wall attachments. If proper decompression has been carried out, all acutely herniated viscera are readily reduced and the laceration of the diaphragm repaired by the placement of interrupted silk sutures (Fig. 11, *B* and *C*).

As time passes, subsequent to the original trauma, reduction of the displaced viscera may become extremely difficult because of "loss of domicile" of the herniated viscera into the chest. If one is operating on a known rupture of many years' duration, it is wise to prepare the patient by introducing a long gastrointestinal decompressive tube. Splenectomy may give a slight amount of room in the upper abdomen. The lowering of the patient's feet and the use of muscle relaxants may also be very helpful in replacing the herniated organs. In the chronic cases also, the surgeon may occasionally experience difficulty in closing the entire rent with the poor atrophic diaphragmatic tissue that remains. Fascia lata as well as various artificial substitutes have been suggested to close the defects. We have found the simple expedient of turning down a piece of pericardium on a pedicle (Fig. 11, *D*) to be quite effective and quite strong in closing a defect in the oldest diaphragmatic rupture (Case 13, D. D.). This pericardial flap can be made of quite large size. It is very strong and appears to fill the needs of a tissue substitute very satisfactorily.

Occasionally, the tightness of the hernial ring makes it impossible to reduce the viscera directly and the enlargement of the hernial ring directly is attended with considerable danger in terms of damage to the herniated viscera. In such instances, we have found counter incisions to be extremely helpful in enlarging the original opening safely and in reducing the herniated viscera without difficulty and without damage to them.

The postoperative care of these patients is usually simple. Within a day of operation, the intrathoracic drainage tube usually can be removed. The nasogastric tube is removed as soon as peristalsis has returned. In general, the patient is out of bed within 24 to 48 hours and out of the hospital within 10 days.

## SUMMARY AND CONCLUSIONS

Traumatic rupture of the diaphragm is becoming a more frequent problem apparently as the result of an increasing number of accidents associated with rapid deceleration.

Sixteen cases of diaphragmatic rupture are reported and an analysis of the types of injury, associated trauma, early and late symptomatology, and findings at surgery is made.

In the acutely severely injured patient, it is understandable that the diagnosis may be readily missed. However, if the possibility is borne in mind, the condition can usually be suspected, diagnosis made early, and prolonged disability avoided.

It is important that the patient have a diaphragmatic injury diagnosed and treated at the time of original hospitalization, in order to obviate the possibility of a later emergency operation, and to assure to him the maximum insurance benefits to which he may be entitled.

The clinical picture of diaphragmatic rupture may be conveniently divided into 3 phases, an acute phase, a chronic phase, and an intermediate or dramatic phase. In the acute phase, the patient is often seriously injured and may have the diaphragmatic injury masked by other serious concomitant trauma. In the chronic phase, the diagnosis may be confused with vague upper abdominal chronic gastrointestinal disease. In the intermediate or dramatic phase, the diagnosis gives the symptoms of acute high or low intestinal obstruction. In this late and dramatic phase, emergency surgical therapy may have to be carried out under less than ideal circumstances.

Diagnostic measures for both the early and late types of diaphragmatic rupture are described, a technical approach to the lesion is suggested, and a maneuver of closing a persisting diaphragmatic defect with a pedicle pericardial flap is described.

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## DISCUSSION

DR JOHN SNYDER, Bethlehem, Pa.—My personal experience has been limited to 4 cases, 3 of the acute type and one chronic. I think one of these of the acute type shows some of the interesting problems under discussion, and I would like to show it.

The patient was a 6-month-old male who was being carried in his mother's arms in an automobile which was forced into the path of an oncoming car, the car being struck on the right side. The mother suffered multiple rib fractures, a pneumothorax and, on admission to the dispensary, the child appeared drowsy but at that time showed no other external

marks of violence or injury. An x-ray was taken and showed a long linear fracture of the right parietal bone with separation of the fragments of about 2 mm. in the x-ray. Despite the absence of dyspnea or abdominal findings a routine chest x-ray was taken.

(slide) You see the radiolucent shadow at the lateral border of the heart. This was taken about 2 o'clock in the morning and the intern called to say that the infant had a localized pneumothorax. He was instructed to pass a Levin tube and take another film.

(slide) This then showed reduction in size and confirmed the diagnosis that we were dealing with a ruptured diaphragm, with the stomach in the chest. The problem then became one of determining the optimum time for operative reduction, in view of the fractured skull. The Levin tube was left in place and the following morning the gastric shadow was no longer visible but you see some other shadows in the lower part of the chest. Fluids were administered by subcutaneous clysis and the cerebral status seemed to be fairly well stabilized. The problem of further fluid administration was rather difficult, however, as the resident staff was having trouble with the veins, and some fluid was given by mouth.

(slide) About 6:00 P.M. the patient became suddenly dyspneic and cyanotic, and you see the stomach now fills the entire left chest. The Levin tube was again replaced.

(slide) You now see that the place where the stomach had been is now largely occupied by coils of bowel. About 4:00 A.M. next morning the child had a convulsion, but 4 hours later seemed to be in relatively good shape. He had developed a moderate hematoma over the fracture site, the fontanelles were not bulging, and it was decided that we could go ahead with repair. This was carried out through the left subcostal region. The rent in the diaphragm was approximately 2 inches in length, began at the left side of the triangular ligament of the left lobe of the liver, continuing in a lateral direction. We first did a phrenic crush in the neck, after which the abdominal repair was accomplished without difficulty, using silk sutures. The child's convalescence was uneventful.

(slide) Two weeks later, prior to discharge, barium was given by mouth, which demonstrated the stomach in normal position.

Acute gastric dilatation in the intrathoracic stomach has been a problem in all 3 of the acute cases I have seen. Fortunately, all have recovered satisfactorily. One other case in this group became a problem because of some peculiar angulation of the stomach, and the Levin tube did not seem to reduce the dilatation of the stomach. An emergency thoracotomy was necessary and a gastrotomy was performed for deflation, after which the diaphragmatic rent was repaired and subsequent recovery was very satisfactory.

(Reproductions of slides are available if desired and usable. [J.S.])

DR. J. W. BROOKS, Richmond, Va.—I have enjoyed this timely discussion of an interesting problem and I would like to add one additional tool in the early diagnosis of these cases.

We have recently been interested in making the diagnosis in the really acute phase as soon as possible, in order to prevent some of the late complications and, in an effort to do so, have utilized a diagnostic pneumoperitoneum. With the production of diagnostic pneumoperitoneum in an acute rupture of the diaphragm, a pneumothorax will be formed on the side of rupture provided the air goes through the foramen. In 4 cases of traumatic rupture of the diaphragm during the last 12 months we have made the diagnosis in 3 by the production of pneumoperitoneum resulting in pneumothorax.

DR. ROBERT M. JANES, Toronto, Ont.—I have not anything very important to say. I was interested in this paper and as I listened to it it reminded me of what to me is a rather interesting historical episode in my own life. Many years ago when my old chief, Dr. Gallie, was chiefly interested in finding a new way to use fascia lata, a patient with a traumatic rupture of the diaphragm came in. The chest was then, to most general surgeons, completely forbidden territory; therefore I opened the chest, he put in the fascia lata to repair the hernia—which was his only interest in the problem—and I closed the chest. I subsequently reported that case and, so far as I know, it was the first reported

incidence of repair of a diaphragm with fascia lata. As Dr. Harrington has so often pointed out, in wide defects that are long standing and which are complicated by a lot of fibrosis, it is still a good method of repair.

There is just one other thing I would like to say, that is, that in the patient who is admitted in extremis when it is obvious that he may die of dyspnea, and one cannot be certain whether he is dealing with a compression pneumothorax or acute dilatation of the stomach as a result of injury, it is not such a terrible thing to do a transthoracic aspiration, if you must, and save the patient's life.

DR. JOSEPH L. LUCIDO, St. Louis, Mo.—We have a series of 25 cases of rupture of the diaphragm due to external violence. Twenty-four were caused by automobile accidents. It is obvious that the cars travel just as fast west of the Mississippi River as on the East Coast. A consistent location of the tear in the diaphragm has been observed; namely, radial in direction and directed anterolaterally from the left crus.

(slide) As this indicates, 23 have been on the left and 2 on the right side. Nineteen patients have been operated upon, with 13 survivors. Six patients on whom operation was not performed have died. The associated major injuries have been rupture of the spleen in 7 cases, severe head injuries in 9 patients, multiple severe fractures of the pelvis in 8 cases, fracture of the femur in 7, and laceration of the pericardium in 3. The laceration of the pericardium has been a pleural-pericardial extension of the diaphragmatic tear, along the same line. These 3 patients survived operation. Most of the deaths in our series have been attributed to the head injuries and the intracranial hemorrhage.

DR. FREDERICK G. KERGIN, Toronto, Ont.—Dr. Desforges has already referred to the fact that in some of these patients who have sustained the rupture a long time before, the defect may be large and cause difficulty in closure, and he suggests using a flap of pericardium as a patch. I think at times the defect may be very large indeed, and even too large for that maneuver. We have had one such problem in which we had to try to find a solution.

(slide) This man of 42 years of age had been injured 15 years before and you see that most of the movable organs of the abdomen are in the left chest. At operation through the chest, the diaphragm was reduced to a narrow rim of tissue with a very large defect in the center. We were misled by the extreme degree of relaxation obtained by the paralyzing drug and attempted direct repair with silk sutures. This patient happened to be a person who struggled violently coming out of the anesthetic, and it was soon obvious that the hernia had recurred. At reoperation a week later, it was found that he had ruptured the diaphragm along a new line.

(slide) In order to close the very large defect now present, we used stainless steel wire mesh. It proved very satisfactory indeed, and the patient has remained well. I think this might prove a useful measure for very large defects.

DR. GERARD DESFORGES, Boston, Mass. (Closing).—I should like to thank the discussors for their remarks. Dr. Snyder pointed out that, in one case, decompression could not be effected by a Levin tube. This does not seem to be an unusual finding. For this reason we feel that if there is a progressively enlarging stomach in the chest, and a diagnosis of rupture has been made, one should proceed with operation immediately. The idea of pneumoperitoneum as an aid to diagnosis has occurred to us. However, in general, the rupture on the left is easily diagnosed. On the right, because of the usually abnormal position of the liver in this condition, we have discussed the use of intravenous cholangiographic dye to outline the biliary tree in its intrathoracic position. The suggested repair with fascia lata or stainless steel mesh I think is excellent if one finds a condition where other material must be employed, and that can be decided at the time of operation.

I would like to say one thing about the position of the tear. As has been pointed out by Dr. Harrington, it can occur anywhere.

# SUPERNUMERARY INTRATHORACIC RIB

## A Case Report

George E. Plum, M.D.,* Alvin B. Hayles, M.D.,** André J. Bruwer, M.B., Ch.B.,*** and O. Theron Clagett, M.D.****
Rochester, Minn.

> Eve was made from the thirteenth rib of the right side. . . . She was not drawn out by the head lest she should be vain; nor by the eyes, lest she should be wanton; nor from the mouth, lest she should be given to garrulity; nor by the ears, lest she should be an eavesdropper; nor by the hands, lest she should be intermeddling; nor by the feet, lest she be a gadder; nor by the heart, for fear she should be jealous; but she was taken out from the side. Yet in spite of all these precautions she had all the faults so carefully provided against. (Quoted from Rabbi Jonathan's Targum, by G. W. Niven, Brit. M. J. 2: 1732, 1898.)

A WHITE girl, 9 years of age, was brought to the Mayo Clinic in November, 1955, with a chief complaint of "lung tumor." The patient had no symptoms referable to the thorax; but in a roentgenogram made by a mobile unit on July 14, 1955, a well-defined area of increased density had been seen adjacent to the hilus of the right lung and extending from the level of the anterior end of the second rib to the right cardiophrenic angle. Roentgenograms measuring 14 by 17 inches had been taken and additional investigations, including cardiac studies, had been advised. The patient had been hospitalized elsewhere, but further roentgenologic studies, including roentgenoscopic observation as barium was swallowed, electrocardiograms, and examination of the blood, had not identified the intrathoracic mass. However, the examiners had considered that the involvement was pulmonary, not cardiac. Surgical exploration had been advised, whereupon the patient had come to the Mayo Clinic for further examination.

In the history or on physical examination evidence of anything abnormal was not found. In roentgenograms of the thorax, made in posteroanterior, lateral, and oblique projections, was a shadow with a convex lateral border lying far posteriorly on the thoracic wall (Fig. 1, *a* and *b*). It appeared to originate near the right lateral border of the sixth thoracic vertebral body and extended downward posteriorly to the ninth intercostal space. The cardiac shadow was of normal size and contour. Twelve pairs of normal ribs were present. Diagnoses that were considered included anomalous blood vessel, intrathoracic cyst, lipoma, and duplication of the esophagus. The electrocardiogram, results of the tuberculin skin test, and examinations of blood and urine all gave negative results.

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Exploratory thoracotomy was performed. An anomalous supernumerary rib lay within the thoracic cage but outside the parietal pleura. The rib originated from the right lateral margin of the sixth thoracic vertebra, anterior to

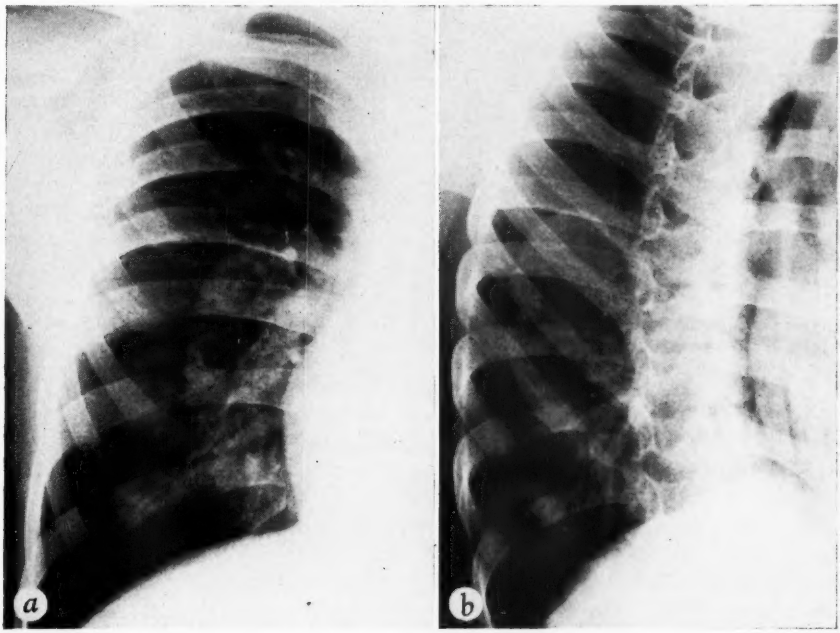


Fig. 1.—*a*, Posteroanterior roentgenogram showing the shadow cast by the supernumerary intrathoracic rib on the right side. *b*, Right anterior oblique view.

Fig. 2.—Resected gross specimen of supernumerary intrathoracic rib.

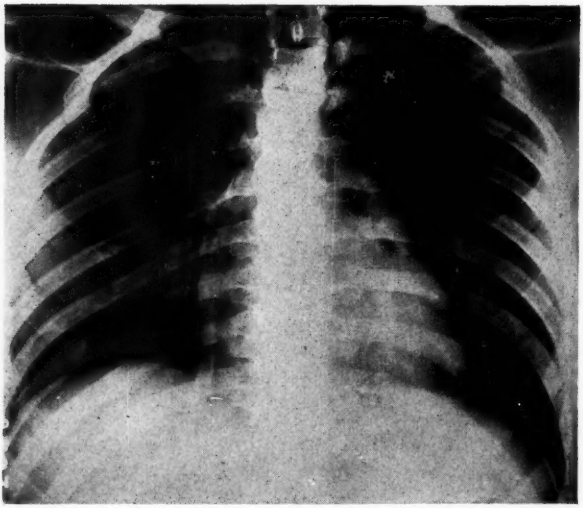

Fig. 3.—Roentgenologic appearance of the intrathoracic rib in the patient described by Marbut and DeGinder. (Reproduced with permission.[2])

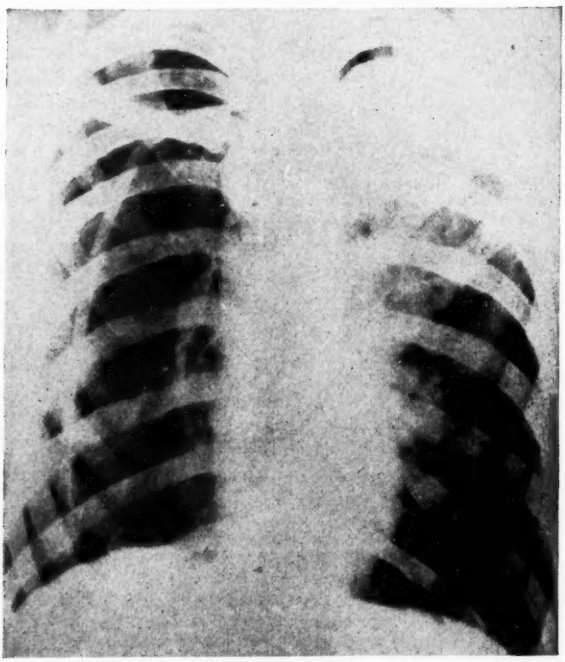

Fig. 4.—Roentgenogram showing the shadow of the rib in Jacobs' patient. (Reproduced with permission.[1])

and between the vertebral articulations of the sixth and seventh ribs; and thence it extended downward posteriorly to about the tenth rib. It was completely removed without difficulty (Fig. 2).

## PREVIOUS REPORTS

Anomalous supernumerary intrathoracic rib is rare and difficult of diagnosis. As far as we know, this is the fourth such case to be reported, and the second in which surgical removal was performed.

Marbut and DeGinder reported on a similar supernumerary rib of a woman, 20 years of age. It was in the same location as the one reported here, and had a similar roentgenographic appearance (Fig. 3).

Sydney Jacobs reported that a supernumerary intrathoracic rib was removed at necropsy from the body of a Negro who was 43 years of age at death. The rib had originated from the right side of the third thoracic vertebra on its anterolateral surface, articulating independently of the normal rib, and extended downward to the level of the eighth thoracic vertebra (Fig. 4). It lay on the anterior surface of the normal ribs, external to the pleura.

Stevenson and Merendino reported their surgical findings in the case of a schoolgirl, 17 years of age, who had two supernumerary ribs on the right side. They were at the levels of the sixth and seventh thoracic vertebrae and extended from their articulations on the vertebral column inferiorly and ventrally for 8 and 11 cm., respectively. At their terminations they projected into the thoracic cavity 1.5 and 2.0 cm. beyond the normal contour of the thoracic wall. Their exposed surfaces were covered with parietal pleura. The right hemidiaphragm was tented upward and was attached to the medial surfaces of these ribs at the level of the sixth and seventh thoracic vertebrae. The patient also had a cervical rib on the right side. The authors cited publications which offer a possible explanation for the abnormal embryologic development in this case.

## DISCUSSION

We have no criteria to offer in making a roentgenologic diagnosis of supernumerary rib. All ribs of the type with which we are concerned here have occurred on the right side. In some cases they evidently were composed of bone such as that of which a rib ordinarily is composed. In our case, the roentgenologic identity apparently was masked by the pleural reflections from the rib; these imparted a "soft" cystlike appearance. Possibly use of tomography would have clarified the nature of the shadow.

On a routine posteroanterior thoracic roentgenogram, the shadow of an anomalous right pulmonary vein might be confused with that of an anomalous rib such as has been described. Usually, however, on routine films or on tomograms, it is possible to see evidence of the branching of anomalous vessels.

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# LONG-STANDING THROMBOSIS OF THE PULMONARY ARTERIES COMPLICATING VALVULOTOMY FOR MITRAL STENOSIS

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THE large number of mitral valvulotomies now being performed brings new importance to the rare complication of mitral stenosis usually described as "massive thrombosis of the pulmonary arteries." This condition leads ultimately to death from failure of the circulation and it has been of academic interest until recently when its existence must be taken into account in evaluating the indications for surgery. As the right pulmonary artery is affected much more commonly than the left, exposure of the heart through a left thoracotomy would be disastrous unless special efforts were made to keep the left lung ventilated.

This paper describes 3 cases of mitral stenosis in which circulatory collapse during or just after operation was later discovered to be due to long-standing occlusion of the right pulmonary artery. It is of interest that 2 patients survived the operation but died shortly afterward from cerebral embolism. The pathologic significance of these massive occlusions is discussed as well as the clues that may lead to preoperative diagnosis.

## CASE REPORT

CASE 1.—The patient, a 27-year-old woman, had had rheumatic fever as a child and had suffered from symptoms of cardiac insufficiency from that time. Three months before death she had a bout of substernal pain radiating to both arms, accompanied by severe dyspnea. The dyspnea gradually increased in severity. On examination three weeks before death, she was dyspneic at rest and slightly cyanosed. The heart was enlarged. Aortic and mitral, systolic and diastolic murmurs could be heard. The pulse rate was 100 per minute and blood pressure was 110/85 mm. Hg. She had several bouts of severe shortness of breath associated with wheezing and cyanosis. The hemoglobin was 80 per cent and red cells were 5.5 million per cubic millimeter.

Eighteen hours before death a mitral commissurotomy was performed. Shortly after the chest was opened, the blood pressure fell to shock levels and remained low for twenty minutes. The valve was split satisfactorily. Postoperatively the patient recovered from the anesthetic and appeared to be doing well. Three hours later she became cyanosed and went into shock. The left arm became flaccid and the right spastic. She remained cyanosed and in shock for the last few hours of her life.

At necropsy, the right pulmonary artery was almost completely occluded by a firm laminated thrombus which was adherent to the wall of the artery. Microscopically, it was demonstrated that the thrombus was being organized. It was estimated to have been present for several weeks, perhaps dating back to the episode of substernal pain three months before death. The right lower lobe showed some interstitial fibrosis but no evidence of infarction. Severe endarteritis of the pulmonary arterioles was present throughout both lungs. The mitral valve was calcified and stenosed, admitting one finger. The

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lateral commissure had been split for 1 cm. and the medial for 0.2 cm. A large thrombus was present in the left auricle. There were thickening and calcification of the aortic valve. The left auricle was dilated and the right ventricle hypertrophied. A small recent infarct was present in the interventricular septum. The heart weighed 405 Gm., the left ventricle was 1.7 cm. and the right 0.6 cm. in thickness. No thrombi were found in the peripheral veins. There was no microscopic evidence of active inflammation nor rheumatic fever in the heart or pulmonary artery. The small cardiac infarct and the cerebral symptoms had been caused by emboli.

CASE 2.—A 43-year-old housewife first had symptoms of cardiac insufficiency at the age of 31 during her second and last pregnancy. These symptoms gradually increased in severity so that shortly before death she was only able to walk one block without shortness of breath. For the last three years of her life she had been having attacks of paroxysmal nocturnal dyspnea. For the six weeks prior to her death her condition had been considerably worse.

Examination on admission revealed that she was failure free. The heart was not enlarged. There was an apical diastolic thrill associated with a rumbling diastolic murmur. The pulse rate was 76 and the blood pressure 110/75 mm. Hg.

Forty-eight hours before death a mitral commissurotomy was performed. Shortly after the patient was turned on her side and the chest opened, the blood pressure was found to be 60/0 mm. Hg and it remained low during most of the operation despite vigorous attempts to raise it. The left auricular appendage was found to contain thrombus adherent to the wall. The lateral commissure was calcified. It was possible to split the medial one by finger fracture while the lateral had to be cut. Postoperatively, she did not regain consciousness and was found to have a left hemiparesis.

At necropsy, there were multiple cerebral softenings probably resulting from emboli. Partially occluding the right pulmonary artery and extending into the branches of the lower lobe, there was a firm, white, laminated thrombus. This was firmly adherent to the wall of the artery and microscopically was found to be undergoing organization. It was estimated to have been present for four to six weeks. There was no evidence of infarction of the lungs. Both lungs showed severe endarteritis and moderate atheroma of the arteries. Old and recent infarcts were found in the kidneys. The heart weighed 435 Gm., the left ventricle measured 1.7 cm. and the right 0.6 cm. There was no evidence of active rheumatic fever in heart muscle, valves, or pulmonary artery. A moderate number of fatty and fibrous plaques of atheroma were present in the big branches of the pulmonary artery.

CASE 3.—The patient, a 38-year-old woman, had had rheumatic fever when she was 8 years of age and started to have dyspnea when she was 23. Three years later she had a pulmonary embolus in the late stage of a pregnancy and shortly afterward the symptoms and signs of a myocardial infarct appeared. Symptoms of another pulmonary embolus followed hysterectomy eighteen months before death. Mitral commissurotomy was performed to try and relieve her increasing dyspnea. After the operation she became blue when the intratracheal tube was removed and died twenty minutes later. At necropsy she was found to have long-standing total occlusion of the pulmonary artery to the right lower lobe. There was a very old white infarct of the lower half of this lobe, another white infarct of lesser age in the upper half of the lower lobe and a fresh hemorrhagic infarct of the lower half of the right upper lobe. The right pulmonary artery was enormously distended and measured 3 cm. in diameter and was very atheromatous. On the left side many of the small branches were occluded with clot, there were two recent localized infarcts with base about 2 cm. in diameter and the main pulmonary artery was occluded with a recent red thrombus. Microscopically, this was found to have a few invading capillaries at its periphery and so it had been present at the time of operation. This woman was for practical purposes existing on part of the right upper lobe and she had not enough reserve to survive thoracotomy. A film of this woman's chest had been taken at the time of her hysterectomy. This was made available to us and in it could be recognized a shadow cast by the dilated right pulmonary artery.

SUMMARY OF CASE REPORTS

The similarity between the first 2 cases is striking. Both were middle-aged women with long histories of cardiac insufficiency from rheumatic mitral disease. Both had severe cellular thickening of the pulmonary arterioles indicative of long-standing pulmonary hypertension. Neither had suffered any sudden episode suggestive of an embolism of a main pulmonary artery but Case 1 had had lesser symptoms of pulmonary embolism three months before death.

Both suffered a severe drop in blood pressure when the chest was opened, cyanosis and low blood pressure persisted after operation, both died later from cerebral embolism, and both were found at necropsy to have almost complete occlusion of the right pulmonary artery by organizing thrombus.

These cases show that immediate survival of operation is possible even when there is almost complete occlusion of one pulmonary artery. The third patient did not survive operation.

*The Significance of Long-Standing Massive Occlusion of the Pulmonary Arteries.*—Before Virchow's time, all clots found in the pulmonary artery were regarded as being due to local thrombosis. Virchow disproved this effectively, and the pendulum swung so far the other way, that one now rarely hears of pulmonary thrombosis. It is understandable, however, that a vast literature accumulated round this subject and there was another spate of papers between 1920 and 1930, when the question of a relative or absolute increase of deaths from thrombo-embolism received the same sort of attention that carcinoma of the lung is enjoying today. Summaries of the forgotten battles are given by Ceelen,[1] and echoes of them keep on cropping up in the English literature. One of the best individual papers on the subject of thrombosis and embolism of the pulmonary artery was written by Møller[2] after a detailed study of 15 cases in which there was massive occlusion of the pulmonary artery and a series of 176 consecutive post mortems in which the arterial tree of the lung was dissected and examined microscopically.

He concluded that although, as Virchow showed, fresh emboli can be distinguished quite easily from thrombi because of their shape and disposition, these features become so altered with age by secondary thrombosis, hyalinization, or organization with incorporation into the vessel wall, that differentiation may be extremely difficult. He described the microscopic clues that emboli are usually, but of course not always, composed of stagnation thrombi whose long fibrin lines run parallel to the walls of the vessel they are formed in but not to the walls of the vessels of lodgment. Pulmonary thrombi on the other hand are composed mainly of platelets and the lines of platelets run at right angles to the vessel wall.

Virchow found furthermore that old emboli may cause distortion of the "spur" at the site of arterial bifurcation. When secondary thrombosis occurs it is usually of "stagnation" type distal to the obstruction and platelet,

"coagulation" type cardial to the obstruction. The important point in differentiation is to take sections for microscopy in the long axis of the vessel and running across the spur of bifurcation.

In his 176 post mortems there were 51 occlusions made up as follows:

| | |
|---|---|
| Embolism without secondary thrombosis | 24 |
| Embolism with limited secondary thrombosis | 13 |
| Embolism with marked secondary thrombosis | 10 |
| Embolism with preponderating thrombosis | 3 |

In the last group the right main artery was affected and all of them occurred in cases of severe mitral stenosis. Careful examination showed all of these thrombi to be at some place continuous with old peripheral emboli.

Møller thought that they had all grown by accretion of thrombus or emboli. He doubted whether the associated pulmonary atherosclerosis was a prime factor in the thrombosis because it was just as severe in the non-thrombosed side. He recognized the presence of inflammatory changes in the vessel wall but considered them to be due to the autolysis of the thrombi. He thought that changes in the composition of the circulating blood could not be of great importance because they would not be solely effective at a localized site such as the right pulmonary artery but that in mitral stenosis the disturbances of blood pressure, blood flow, and vortex formation could obviously play a very important role.

It is obvious that from a study of 176 cases, conclusions cannot be drawn to cover all possible occurrences in the pulmonary artery but as far as the massive occlusions associated with mitral stenosis are concerned we are in agreement with Møller that they are derived by thrombotic overlay on an embolus. Since Møller's observations, little new has been added to the literature. Pou and Charr[3] list the authors who variously think that disease of the blood, vessel wall, or blood flow plays a more important role than previous embolism. A report of cases of thrombus of the pulmonary artery in association with defects of the interatrial septum was made by Canada and associates,[4] who incriminated atheroma and increase of pulmonary blood volume. Both Pou and Charr and Savacool and Charr[5] described cases of thrombosis of the pulmonary artery in severe tuberculosis of the lungs. In tuberculosis, the thrombi extend to areas of gross tissue destruction and form at least one exception to Møller's thesis that thrombi are always related to previous emboli. A survey of the published cases of massive thrombosis of the pulmonary artery shows that very little attention was given by the authors to the limits and structure of the occluding mass, the diagnosis nearly always being presumptive and resting on the fact that an acute episode was lacking in the history. In our material of approximately 10,000 necropsies, we have only 6 coded cases of thrombosis of the pulmonary artery and on critical re-examination all except one could be ascribed to or closely related to previous emboli. Savacool and Charr collected from the literature 88 cases described as primary pulmonary thrombosis. Of these, 21 occurred in cases of mitral stenosis, 3 in

severe pulmonary atheroma, 17 in inflammations of the lung, 12 in heart failure, and 15 in various cachectic states. In the remaining 17 cases, occlusion was limited to lobar branches. One's first feeling would be to eliminate these 17 cases of limited occlusion from the group we are discussing but of course there is no magic in the actual size of the pulmonary artery occluded. It is commonplace to find multiple emboli throughout the branches of the pulmonary artery in mitral stenosis. These may or may not produce pulmonary infarction but the effects of occlusion may cause exactly the same symptoms and side effects as the more dramatic occlusion of the main branch. The important point is how much of the pulmonary circulation has been occluded and, looked at in this way, we may assume that arterial occlusion is probably a far more frequent problem than one would judge from the literature.

One feature of great interest is why the right pulmonary artery is more often affected than the left. This was noticed by Møller who ascribed it to the fact that emboli occur more frequently on the right side. In the cases collected by Savacool and Charr[5] the occlusion was on the right side in 49, on the left in 6, and bilateral in 45. Whatever the reason, the fact of this discrepancy has great importance when the function of the left lung is handicapped by thoracotomy.

The effects of occlusion of the pulmonary artery have not been studied in great detail. Gibbon and co-workers[6] found that in cats obstruction of up to 60 per cent of the cross section of the main pulmonary artery was without effect. When occlusion reduced the lumen by 60 to 85 per cent, the blood pressure of the animals fell and further occlusion was fatal.

## DIAGNOSIS

The problem in diagnosis is not to differentiate between thrombosis and embolism. It is to distinguish between those cases of severe disability with cyanosis, dyspnea, hypertrophy, and dilatation of the right heart, accentuated secondary pulmonary sound and probable right heart failure which are due to simple mitral stenosis and those which are due to stenosis plus the added complication of organic obstruction of the pulmonary arteries. There is help if the disability is far greater than would be expected from the degree of stenosis that is believed to exist, but this is not a very strong position to be in. Our Case 2 had progressive dyspnea but no signs of right heart failure. To the dominant picture of severe dyspnea must be added the effects of chronic and increasing anoxia on the brain causing disturbances of thought, judgment, and emotion. Examination of the lungs reveals fewer signs than would be expected if the symptoms were solely due to mitral stenosis. Radiology may or may not help. A pulmonary artery full of thrombus does not differ much in density from an artery full of blood but Keating and associates[7] and Hanelin and Eyler[8] have reported some changes. These include lack of pulsation and alteration of the contour of the vessel at the site of the obstruction, dilatation proximal to it, and increased radiolucency of the lung due to decreased blood

flow. In our Case 3, the increase in size of the shadow cast by the right pulmonary artery was obvious in retrospect. The pulmonary pressure is high and, in one of the cases reported by Canada and associates,[4] a catheter could not be made to enter into the pulmonary artery. In one of Carroll's[9] cases there was absence of vascular markings in angiocardiograms and this would seem to us the most reliable sign of all.

## PROGNOSIS

This condition is not yet treatable and the life expectancy of patients with occlusion of the main stem is probably short. In our 6 cases of massive pulmonary thrombosis which did not come to operation death occurred 8 weeks, 4 weeks, 4 weeks, 7 weeks, 12 weeks, and 6 weeks, after the onset of severe disabling symptoms, an average of 7 weeks. We doubt if valvulotomy is justified in such cases. If the lesion is diagnosed it must be borne in mind as a cause of profound fall in blood pressure during operation and unusual care should be taken to ventilate the left lung.

## SUMMARY

Three women with mitral stenosis who died shortly after valvulotomy were found to have long-standing occlusion of one or more main branches of the pulmonary artery. Such a condition must be thought of if a patient suffers severe shock when the chest is opened. These massive occlusions occur more commonly on the right side and are thought to be due to accretions of thrombus on top of emboli. Multiple small emboli can produce just as much total occlusion and such a diminution in effective pulmonary circulation would seem to be a contraindication to operations on the heart. Angiography offers the most promising approach to diagnosis.

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# ENTEROGENOUS CYST OF THORAX WITH PANCREATIC TISSUE AS A CONSTITUENT

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ENTERIC cysts of the mediastinum have often been reported and the subject has been reviewed by Fallon, Gordon, and Lendrum[1] who give an excellent account of the embryology, development, and associated anomalies. In these cases the cysts have been lined by ciliated, gastric, or intestinal epithelium. We present here an example in which the lining was of biliary tract type with differentiation to well-formed pancreatic tissue.

## CASE REPORT

*Clinical History.*—A previously healthy girl, aged 15 years, attended a General Hospital in May, 1951, complaining of multiple joint pains, shortness of breath, and night sweats. Chest x-ray revealed an enlarged cardiac shadow with patchy opacities in the lower zones of both lung fields. A large effusion then developed in the right side of the chest and aspiration showed dark brown, slightly viscid fluid containing moderate numbers of degenerate mononuclear cells. Microscopic and cultural examinations of the fluid for microorganisms including tubercle bacilli were negative. The patient's Mantoux reaction was positive.

A rheumatic etiology was considered but tuberculosis seemed more probable. A course of streptomycin and PAS was given with the later addition of isoniazid. She gradually improved and after several months of in-patient treatment was sent home and followed up. Clinically she then appeared to be fairly well, but the enlarged cardiac shadow persisted radiologically and indeed began to increase in size.

In December, 1952, there were further severe bouts of pains in the joints and, also, in the calves on exertion. She was readmitted in March, 1953. Paracentesis of the right chest produced fluid similar to that obtained previously and full bacteriologic examination was again negative. Her condition improved but she continued to have relapses of pyrexia with pains in the joints and legs. The diagnosis now considered was for tuberculous pericarditis and she was referred to the Thoracic Surgery Unit with a view to pericardectomy.

When first seen here she was pale and rather dyspneic with a pulse that was regular but too rapid to count. There was visible pulsation in the neck and epigastrium. After rest the pulse settled at 110 per minute. Heart sounds were not distinct but no murmurs were detected. At the base of the right lung, the percussion note was impaired, with bronchial breathing and moist sounds. Maximum temperature was 99.8° F. Hemoglobin was 13.0 Gm. per cent, W.B.C., 8,000 per c.mm. (differential count normal); and sedimentation rate (Westergren), 38 mm. in one hour.

Aspiration of the right chest in the mid-axillary line yielded 5 pints of greenish-brown viscid fluid. This showed no cholesterol crystals microscopically but demonstrated a moderate number of red blood corpuscles and some degenerate cells, probably endothelial. Full bacteriologic examination, including guinea pig inoculation, was again negative; protein content, 1.9 Gm. per 100 milliliters; no bile pigments; and hemoglobin and hematin were demonstrable spectroscopically.

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Following this there was immediate relief of the joint and muscle pains and she felt much better. Three weeks later she again suffered a relapse. Four pints of fluid were then withdrawn, once more with immediate relief of the pains. Two further aspirations were carried out, once with air replacement (Fig. 1).

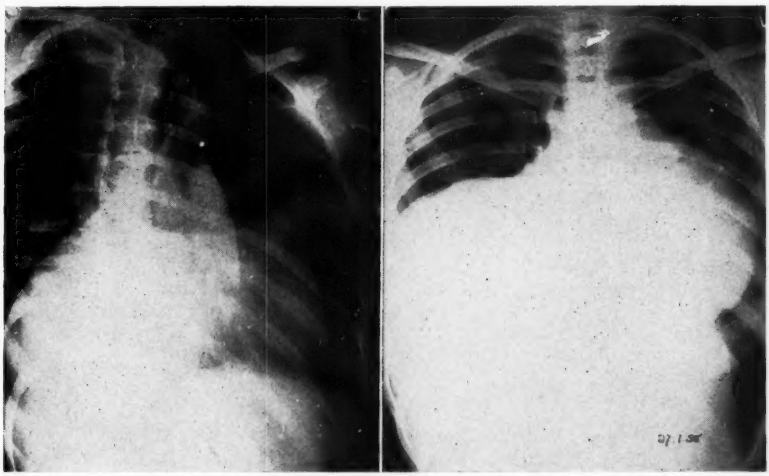

Fig. 1. Fig. 2.

Fig. 1.—X-ray of chest after aspiration of fluid and air replacement.
Fig. 2.—Postoperative x-ray of chest.

Fig. 3.—Postoperative x-ray of chest.

The patient became very tired of hospitals and was anxious to return to work. She, therefore, went home and was lost sight of for a time. She eventually came back. Although her mother said she was exhausted by evening and very short of breath on slight exertion, she herself would not admit it. Operation was advised and accepted. A preoperative x-ray of the chest is shown in Fig. 2.

*Operation—Feb. 18, 1955.*—Immediately prior to operation, a needle was introduced into the right side of the chest and 3½ pints of fluid were aspirated from the pericardium. The patient was then placed in the supine position and an incision was made extending from the posterior axillary line on the right side in the fourth interspace, passing under the right breast across the sternum and under the left breast, to the posterior axillary line on the left side. The breasts were reflected upward and the muscles divided in the line of the fourth interspace. Both sides of the chest were opened and the sternum divided transversely. The left lung and pleural space appeared normal, but the right lower chest

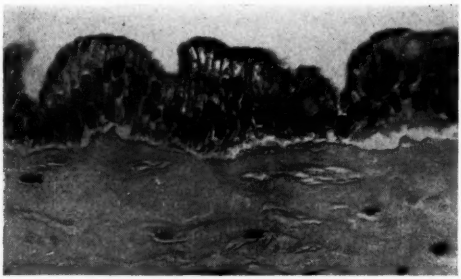

Fig. 4.—Epithelial lining of main cyst wall. (Hematoxylin-eosin stain; ×300.)

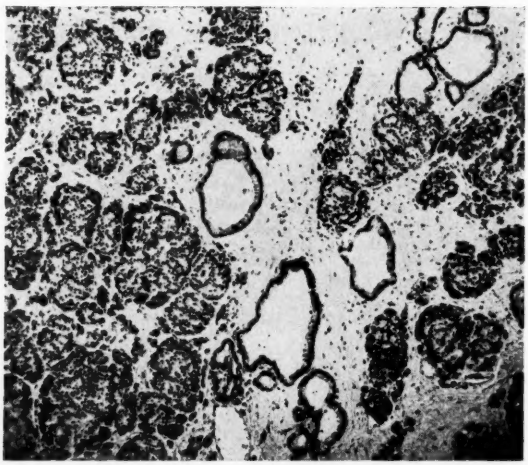

Fig. 5.—Mingled pancreatic acinar and islet tissue together with loculi lined by columnar epithelium. (Hematoxylin-eosin stain; ×60.)

was occupied by a huge tumor mass. As this was freed it was seen to be a large cystic swelling which was confluent with and fused with the pericardium. The cyst was freed by sharp dissection from the lower lobe of the right lung and then from the diaphragm and inferior vena cava. As it was so unwieldly, it was opened and a further 3 pints of fluid removed by suction. The cyst was found not only to be confluent but, also, communicating with the pericardial sac, although the actual communication could not be clearly demonstrated. The cyst wall and pericardium were then freed from the right middle lobe, the right pulmonary artery (to which fusion was very firm), and from the superior vena cava. Then it was noted that a long prolongation passed up over the aortic arch following the innominate artery high in the thorax. This was stripped off and removed with the whole of the pericardium. The cyst wall was about 1.0 cm. in thickness

in places, and had a lining membrane which was thrown up into thick folds similar to gastric mucosa. This lining membrane was noted to be stained deep green. After the cyst had been removed the right lung was freed from the chest wall and completely decorticated. Both phrenic nerves were inextricably involved in the cyst wall and pericardium and had to be sacrificed. The operation took about five hours, and the patient's condition was satisfactory throughout. (A postoperative roentgenogram is shown in Fig. 3.)

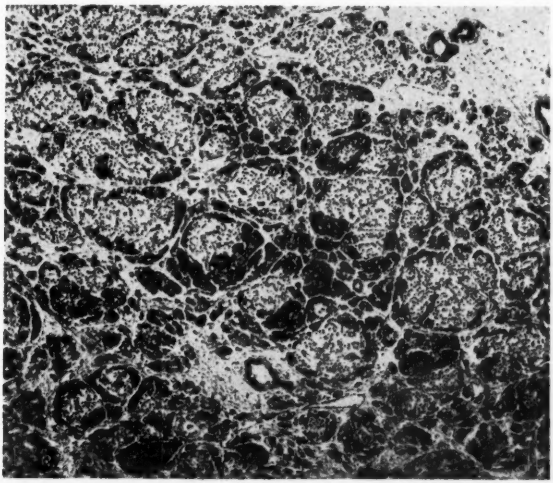

Fig. 6.—General view of mingled pancreatic acinar and islet tissue. (Periodic acid-Schiff stain; ×60.)

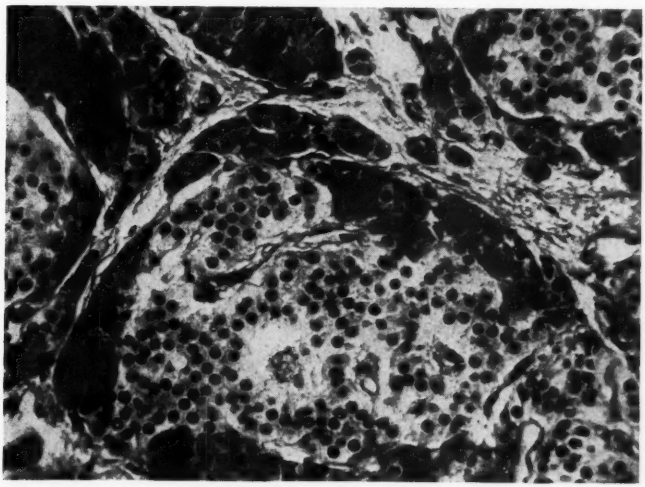

Fig. 7.—Enlarged view to show the distinction between the islet tissue and the heavily stained zymogen-containing acinar tissue. (Periodic acid-Schiff stain; ×300.)

Following operation, the patient had a quite uneventful convalescence. On screening, the diaphragm remains paralyzed but she is completely symptomless and is capable of the most strenuous exertion without undue breathlessness.

*Pathologic Findings.*—The specimen consisted of numerous pieces of pericardium and cyst wall, most of them between 0.5 and 1.0 cm. thick. Attached to one piece was a mass about 5.5 cm. in diameter containing a separate loculus about 2.5 cm. diameter and many other smaller cavities down to microscopic size. Another piece had an attached firm solid nodule about 2.0 cm. in diameter subsequently shown to be thymus. Most of the pieces had a roughened surface but some were fairly smooth.

The lining of the cyst was of tall columnar mucus-secreting cells with basal nuclei (Fig. 4). These gave a positive reaction with Southgate's mucicarmine and periodic acid-Schiff stains, and an alcohol-labile metachromasia with toluidine blue. In places they were somewhat flattened. There were many areas where the epithelium had been shed and replaced by granulation tissue with some chronic inflammatory cells and deposition of hemosiderin giving a positive stain with Perl's reagent, and a little lipofuscin giving negative stains with Gmelin's and Stein's test but positive stains with Schmorl's reaction, Nile blue, and a long Ziehl-Neelsen method (Pearse[3]). The wall consisted of rather hyaline fibrous tissue with a few blood vessels. Most of the arterioles showed endarteritis obliterans.

The loculus and cavities of the mass were lined by cells identical with those of the cyst (Fig. 5). The intervening tissue consisted of loose fibrous tissue, with occasional small bundles of plain muscle, and pancreas (Figs. 6 and 7) in which islet cell tissue predominated and was not only clearly histogenetically related to the acinar tissue, but had, itself, an acinar structure in parts. Some of the islet cells contained red granules when stained with Goodpasture's eosin-methylene-blue and were presumably $\alpha$ cells. Attempts to stain specifically the $\beta$ cells were unsuccessful, as the tissue was fixed in 10 per cent formalin with 2 per cent acetic acid, and these granules had no doubt dissolved. The zymogen granules in the acinar tissue stained a deep purple with Goodpasture's eosin-methylene-blue, dark blue with Mallory's phosphotungstic acid hematoxylin, and red with periodic acid-Schiff stains.

A total of 15 blocks were prepared from the material, but no gastric epithelium or any other tissue was found.

## DISCUSSION

It has been noted that enterogenous cysts are sometimes associated with abnormalities of the spine or posterior ends of the ribs (Fallon, Gordon, and Lendrum[1]), but such abnormalities were not found on radiologic examination of this girl.

An interesting case is described by Löhr[2] of a 10-year-old boy with a cyst in the upper part of the right side of the chest. Fluid from the cyst contained amylase and trypsin. The illustrations show a lining epithelium with occasional glandular acini of rather nondescript type. The author discusses the possibility of the tissue being pancreatic but decides that, "this is as good as developmentally impossible," and concludes that the cyst is parotid tissue. Pancreatic tissue is common in Meckel's diverticula and it is not surprising that a foregut cyst might differentiate in a similar manner.

## SUMMARY

A large cystic lesion in the thorax containing heterotopic pancreatic tissue is described.

Our thanks are due to Professor Willis for the photomicrographs and for his valuable advice and criticism, and to Mr. G. Tulloch for technical assistance.

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# AORTIC COMMISSUROTOMY: A PHYSIOLOGIC EVALUATION BY COMBINED HEART CATHETERIZATION

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FOLLOWING earlier experimental work, the principle of aortic commissurotomy was applied clinically in 1950.[1] During the past six years the techniques for surgical relief of rheumatic aortic stenosis have undergone various modifications.[2-6] In this period, studies of over 400 cases treated by aortic commissurotomy have been possible.[7, 8] While many patients appeared to be improved clinically, it has been most difficult to assess accurately the value of the procedure as applied to patients with aortic stenosis in general.

Prior to the development of left heart catheterization,[9-16] the evaluation of the surgical results has depended upon clinical follow-up for the most part and, in addition, the result of pressure recordings obtained at the time of surgery. While careful clinical follow-up is valuable with a large group of cases, it tends to be somewhat inaccurate and nonspecific with regard to the individual patient. Further, it depends upon subjective as well as objective findings. Many of them are secondary manifestations of the altered hemodynamics and as such lack specific proportion.

Pressure tracings recorded in the operating room before and after commissurotomy reveal more definite information about the dynamics of the lesion. However, this information is influenced by a number of factors inherent in the procedure which render it of limited value. The influence of general anesthesia and vasopressor drugs when combined with the altered physiology of an open chest tend to alter the level of any pressure gradient from that which actually exists during basal conditions. It has been clearly shown by previous studies in our laboratory[17, 18] that the pressure gradient, per se, is not an accurate index of the degree of aortic stenosis since it is a function of the flow across the valve. During most cardiac surgical procedures, blood pressure fluctuation with or without blood loss makes it impossible to maintain the patient in a steady state sufficient to obtain a reliable

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estimation of the cardiac output. Therefore, any measurement of the pressure gradient will be significantly influenced by these factors and of relatively little use in evaluating the result of the valvulotomy.

In this study we have attempted to eliminate the previously mentioned sources of error and have obtained detailed physiologic data by combined heart catheterization on 14 patients both preoperatively and two weeks following aortic commissurotomy.

## MATERIAL

Fourteen patients with pure aortic stenosis were studied, both preoperatively and postoperatively just prior to discharge from the hospital. In every case the diagnosis was confirmed at surgery. All patients were operated upon by the transaortic route. A positive history of rheumatic fever was obtained in 6 cases. However, in all cases except one the lesions were considered to be acquired. The remaining case (M. Z.) was one of congenital subaortic stenosis. The mean age of the group was 43 years with a range of 10 to 62 years. The clinical findings were consistent with the diagnosis of aortic stenosis. Several patients had had recent evidence of congestive heart failure, but were considered compensated by the usual criteria at the time of the catheterization.

## METHODS

The method of study has been detailed elsewhere.[15-17] All patients had a complete history, physical examination, electrocardiogram, and preliminary roentgen studies, including fluoroscopy, performed prior to catheterization. All patients were studied in the postabsorptive state by simultaneous right and left heart catheterization. Premedication of Seconal, 3 grains, was given 45 minutes before the procedure and Demerol, 50 mg., intramuscularly, 30 minutes prior to insertion of the needle into the left atrium. Right heart catheterization was accomplished by the usual technique,[19] except that the patient was postured in the prone position. The catheter was first wedged into the pulmonary artery to record the pulmonary "venous capillary" pressure, then was withdrawn so that the tip lay within the pulmonary artery. A Cournand needle was then introduced into the brachial artery. The left heart needle then was introduced by the transthoracic approach according to the Fisher[14] modification of the Björk technique.[10] A 6-inch, No. 18 gauge, thin-walled needle was inserted under fluoroscopic control after first selecting the site of puncture from the 36-inch posteroanterior chest film. After the left atrium had been

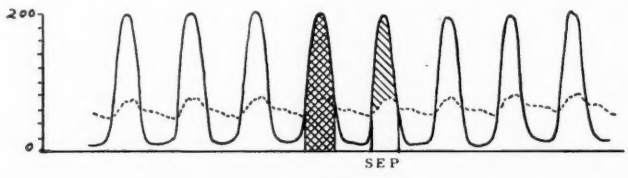



Fig. 1.—Superimposed brachial artery (broken line) and left ventricular pressure (solid line) tracings indicating systolic pressure gradient, left ventricular mean systolic pressure and systolic ejection period.

penetrated, a fine polyethylene catheter was advanced through the needle to enter successively the left atrium, left ventricle and, wherever possible, the aorta. At this point expired air was collected for a 3-minute period in a Tissot spirometer in the middle of which blood samples were simultaneously withdrawn from the brachial and pulmonary arteries. Immediately following removal of the blood samples, tracings were recorded as the tip of the left heart catheter was pulled back across the aortic and mitral valves into the left atrium. On completion of the left heart studies, the right heart catheter was pulled back through the pulmonary artery, right ventricle, and right atrium while continuous pressure tracings were recorded.

All blood samples were analyzed for oxygen content and capacity according to the method of Van Slyke and Neill.[20] Cardiac output was calculated by the direct Fick method. Measurements of left ventricular ejection pressure and systolic pressure gradients were made by planimetry (Fig. 1). Calculations of valve area, flow, and ventricular work were done using a modification of the Gorlin formulae.[21]

Aortic valve area was calculated by the formula:

$$AVA = \frac{AVF}{44.5 \times \sqrt{SPG}}$$

where AVF = aortic valve flow (c.c./S.E.P. sec.)
44.5 = empirical constant
SPG = systolic pressure gradient
i.e., Left ventricular (mean) systolic minus aortic (or brachial artery) mean systolic pressure (mm. Hg).

Aortic valve flow:

$$AVF = \frac{CO}{SEP}$$

where CO = cardiac output (c.c./min.)
SEP = systolic ejection period (sec./min.) (Fig. 1).

Left ventricular total work:

$$LV_{TW} = \frac{(CI \times 1.055)\ (LV_M \times 13.6)}{1{,}000}$$

where CI = cardiac index (L./min./M.$^2$)
$LV_M$ = left ventricular mean systolic ejection pressure (mm. Hg).

Left ventricular stroke work:

$$LV_{SW} = \frac{\text{LV total work}}{\text{Heart rate}}\ .$$

Left ventricular effective work was calculated by the formula:

$$LV_{EW} = \frac{(CI \times 1.055)\ (BA_m \times 13.6)}{1{,}000}$$

$BA_m$ = mean brachial artery pressure
CI = cardiac index.

## RESULTS

The physiologic data obtained from the study of this series of patients are shown in Table I. The pressure gradient across the aortic valve preoperatively ranged from 40 to 75 mm. Hg with an average of 50 mm. Hg. Postoperatively there was a drop in every case except 2 (M. Z. and S. B.) in whom a slight increase was observed. The average decrease in pressure gradient for the 12 patients was 25 mm. Hg with a range of 5 to 45 mm. Hg

TABLE I. AORTIC STENOSIS—

| PATIENT | | OXYGEN CONSUMPTION (C.C./MIN./M.²BS) | ARTERIOVENOUS OXYGEN DIFFERENCE (VOL. %) | CARDIAC OUTPUT (L./MIN.) | CARDIAC INDEX (L./MIN./M.²BS) | HEART RATE (BEATS/MIN.) | STROKE VOLUME (C.C./BEAT) | PULMONIC CIRCULATION | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | | PRESSURES MM. Hg | | | | | |
| | | | | | | | | RIGHT ATRIUM (MEAN) | RIGHT VENTRICLE S/D | PULMONARY ARTERY S/D (MEAN) | PULMONARY CAPILLARY (MEAN) | PULMONARY VASCULAR RESISTANCE (DYNES. SEC. CM.⁻⁵) | RIGHT VENTRICULAR WORK (KG./M./MIN.) |
| S. F. | Pre-op. | 110 | 4.7 | 4.2 | 2.4 | 95 | 44 | 0 | 20/5 | 21/10 (15) | 8 | 114 | 0.38 |
| | Post-op. | 136 | 5.2 | 4.6 | 2.6 | 116 | 40 | 4 | 30/8 | 28/8 (18) | 8 | – | 0.67 |
| J. L. | Pre-op. | 112 | 6.3 | 3.4 | 1.8 | 135 | 25 | 5 | 25/7 | 25/10 (18) | 10 | 141 | 0.47 |
| | Post-op. | 101 | 4.6 | 4.0 | 2.1 | 138 | 29 | 12 | 40/5 | 40/15 (20) | 16 | 100 | 0.60 |
| A. D. | Pre-op. | 116 | 4.8 | 3.7 | 2.4 | 100 | 37 | 12 | 31/11 | 30/18 (20) | 10 | 216 | 0.69 |
| | Post-op. | 136 | 4.7 | 4.6 | 3.0 | 110 | 42 | 10 | 36/12 | 35/17 (28) | 10 | 314 | 1.21 |
| M. M. | Pre-op. | 106 | 5.5 | 3.4 | 1.9 | 85 | 40 | – | – | 32/18 (25) | 11 | 235 | 0.68 |
| | Post-op. | 108 | 5.3 | 3.4 | 2.0 | 105 | 33 | 8 | 23/8 | 27/12 (18) | 9 | 142 | 0.51 |
| S. B. | Pre-op. | 128 | 5.3 | 3.9 | 2.4 | 90 | 44 | 9 | 39/8 | 30/15 (20) | 9 | 249 | 0.69 |
| | Post-op. | 108 | 4.8 | 2.3 | 1.5 | 100 | 23 | 8 | 36/12 | 30/14 (18) | 8 | 346 | 0.39 |
| J. S. | Pre-op. | 108 | 7.5 | 2.7 | 1.5 | 95 | 28 | 11 | 65/14 | 60/35 (50) | 30 | 738 | 1.08 |
| | Post-op. | 149 | 6.8 | 3.9 | 2.2 | 110 | 36 | 13 | 65/10 | 60/30 (40) | 15 | 452 | 1.26 |
| L. H. | Pre-op. | 115 | 3.7 | 5.6 | 3.1 | 90 | 60 | – | – | 30/10 (18) | 10 | 171 | 0.80 |
| | Post-op. | 109 | 4.3 | 4.4 | 2.5 | 98 | 42 | 4 | 35/7 | 35/15 (23) | 11 | 273 | 0.82 |
| J. M. | Pre-op. | – | 5.9 | – | – | 79 | – | 0 | 33/5 | 35/20 (25) | 10 | – | – |
| | Post-op. | 131 | 4.1 | 6.1 | 3.2 | 89 | 69 | – | – | 44/22 (30) | 20 | 183 | 1.38 |
| J. J. | Pre-op. | 137 | 5.3 | 4.5 | 2.6 | 102 | 25 | 4 | 40/4 | 40/10 (22) | 22 | – | 0.75 |
| | Post-op. | 100 | 4.3 | 3.8 | 2.3 | 104 | 37 | 3 | 33/10 | 30/16 (23) | – | 274 | 0.76 |
| C. McD. | Pre-op. | 117 | 6.6 | 3.2 | 1.8 | 76 | 42 | 4 | 27/4 | 27/14 (21) | 11 | 274 | 0.92 |
| | Post-op. | 134 | 5.3 | 4.4 | 2.5 | 75 | 59 | 10 | – | 40/20 (30) | 15 | 182 | 0.08 |

*Measured by planimetric integration.

PHYSIOLOGIC DATA PRE- AND POSTOPERATIVE

| SYSTEMIC CIRCULATION | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| PRESSURES MM. Hg | | | | | | | | | | | |
| LEFT ATRIUM (MEAN) | LEFT VENTRICLE S/D | AORTA S/D (MEAN) | BRACHIAL ARTERY S/D (MEAN) | LEFT VENTRICULAR EJECTION (MEAN) | SYSTOLIC EJECTION PERIOD (SEC./BEAT) | LV—BA GRADIENT (MM. Hg)* | AORTIC VALVE FLOW (C.C./S.E.P. SEC.) | AORTIC VALVE AREA (CM.²) | LEFT VENTRICULAR WORK, EFFECTIVE (KG./M./MIN.) | LEFT VENTRICULAR WORK, TOTAL (KG./M./MIN.) | LEFT VENTRICULAR STROKE WORK (GM./M./MIN.) |
| 9 | 230/6 | 110/78 (86) | 120/72 (84) | 177 | .28 | 74 | 159 | 0.4 | 2.69 | 6.10 | 14 |
| 20 | 200/20 | 130/74 (90) | 120/80 (90) | 142 | .20 | 56 | 197 | 0.6 | 3.37 | 5.12 | 44 |
| 12 | 200/10 | – | 110/80 (96) | 156 | .22 | 52 | 115 | 0.4 | 2.51 | 4.05 | 30 |
| 16 | 170-190/12-12 | 110/80 (90) | 110-120/80-80 (90) | 166 | .20 | 43 | 145 | 0.5 | 2.75 | 5.02 | 37 |
| 10 | 216/19 | 108/60 (70) | 120/70 (90) | 182 | .22 | 71 | 167 | 0.5 | 3.10 | 6.28 | 63 |
| 10 | 160/16 | 99/70 (80) | 118/76 (98) | 147 | .20 | 38 | 209 | 0.8 | 4.25 | 6.35 | 58 |
| 15 | 190/20 | 120/90 (105) | 140/100 (110) | 179 | .26 | 43 | 143 | 0.5 | 2.99 | 4.89 | 58 |
| 12 | 106/12 | 106/77 (90) | 102/72 (80) | 100 | .20 | 0 | 162 | – | 2.28 | 2.86 | 27 |
| 14 | 232/20 | 140/84 (100) | 140/82 (105) | 193 | .24 | 57 | 193 | 0.5 | 3.61 | 6.65 | 65 |
| 8 | 180/18 | 110/70 (80) | 110/74 (84) | 149 | .25 | 68 | 93 | 0.3 | 1.80 | 3.20 | 32 |
| 27 | 162/29 | – | 101/70 (90) | 140 | .28 | 52 | 100 | 0.3 | 1.94 | 3.02 | 37 |
| 18 | 160/20 | 100/60 (78) | 100/60 (80) | 144 | .25 | 47 | 143 | 0.5 | 2.52 | 4.51 | 41 |
| 20 | 180/22 | 120/100 (110) | 130/90 (110) | 166 | .27 | 43 | 232 | 0.8 | 4.88 | 7.42 | 77 |
| 8 | 140/10 | 110/80 (100) | 130/80 (100) | 132 | .19 | | – | – | 3.65 | 4.70 | 48 |
| 14 | 196/16 | 104/82 (96) | 130/80 (92) | 167 | .28 | 50 | – | – | – | – | – |
| 16 | 170/14 | – | 144/80 (98) | 152 | .27 | 12 | 286 | 1.9 | 4.53 | 6.97 | 78 |
| 22 | 220/25 | – | 110/60 (74) | 161 | .32 | 74 | 138 | 0.4 | 2.76 | 6.25 | 60 |
| 10 | 130/18 | 90/60 (70) | 100/60 (70) | 109 | .23 | 29 | 165 | 0.7 | 2.30 | 3.60 | 34 |
| 11 | 210/26 | 140/80 (98) | 142/80 (94) | 165 | .32 | 55 | 131 | 0.4 | 2.43 | 4.26 | 56 |
| 20 | 200/20 | – | 120/60 (86) | 134 | .32 | 43 | 183 | 0.6 | 3.08 | 4.80 | 63 |

TABLE I—

| PATIENT | | OXYGEN CONSUMPTION (C.C./MIN./M.²BS) | ARTERIOVENOUS OXYGEN DIFFERENCE (VOL. %) | CARDIAC OUTPUT (L./MIN.) | CARDIAC INDEX (L./MIN./M.²BS) | HEART RATE (BEATS/MIN.) | STROKE VOLUME (C.C./BEAT) | PULMONIC CIRCULATION | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | | PRESSURES MM. Hg | | | | PULMONARY VASCULAR RESISTANCE (DYNES. SEC. CM.⁻⁵) | RIGHT VENTRICULAR WORK (KG./M./MIN.) |
| | | | | | | | | RIGHT ATRIUM (MEAN) | RIGHT VENTRICLE S/D | PULMONARY ARTERY S/D (MEAN) | PULMONARY CAPILLARY (MEAN) | | |
| M. Z. | Pre-op. | 143 | 5.9 | 4.0 | 2.5 | 96 | 42 | 1 | 18/5 | 22/15 (20) | 14 | 40 | 0.72 |
| | Post-op. | 90 | 5.6 | 2.4 | 1.5 | 102 | 24 | 9 | 25/10 | 28/15 (18) | 18 | 200 | 0.39 |
| H. K. | Pre-op. | 135 | 4.5 | 5.6 | 3.0 | 90 | 62 | 3 | 30/5 | 28/10 (15) | 6 | 129 | 0.64 |
| | Post-op. | 130 | 4.4 | 5.5 | 3.0 | 100 | 55 | 10 | 35/10 | 35/15 (20) | 20 | – | 1.15 |
| W. R. | Pre-op. | 136 | 4.9 | 5.2 | 2.8 | 100 | 52 | 0 | 20/4 | 15/4 ( 8) | 2 | 31 | 0.32 |
| | Post-op. | 143 | 4.5 | 5.9 | 3.2 | 99 | 60 | 6 | 28/0 | 25/10 (15) | 8 | 970 | 0.69 |
| B. S. | Pre-op. | 112 | 5.3 | 3.8 | 2.1 | 95 | 40 | 5 | 26/2 | 20/10 (14) | 14 | – | 0.42 |
| | Post-op. | 110 | 4.4 | 4.5 | 2.5 | 97 | 47 | 3 | 25/8 | 20/9 (10) | – | 354 | 0.36 |

(Fig. 2). Two cases (L. H. and M. M.) showed complete absence of pressure gradient postoperatively (Fig. 3). One of the 2 patients who showed a slight increase in gradient (M. Z.) had subaortic stenosis.

The calculated valve areas of the group preoperatively averaged 0.5 cm.² with a range of 0.3 to 1.1 cm.² Since a normal aortic valve area is considered to be approximately 3 cm.², there was a severe degree of stenosis in all cases but one. The patient with only moderate aortic stenosis (H. K.) with a valve area of 1.1 cm.² had coexisting essential hypertension.

Postoperative data indicated that the functional valve areas had been increased in 11 patients of the group. Of the 3 patients who showed no increase in valve area, one had a subaortic stenosis while another was the individual with coexisting systemic hypertension with only a moderate stenosis preoperatively. The average increase in valve size in those cases where it could be calculated was 0.3 cm.², excluding the 2 patients in whom the gradients were abolished and in whom, therefore, the valve area could not be calculated. The latter is due to a limitation in the formula which requires the existence of some gradient in order to be effective mathematically. Since the gradient was abolished in each of these 2 cases, it is reasonable to believe the functional valve areas were markedly increased and approached normal.

CONT'D

| SYSTEMIC CIRCULATION | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| PRESSURES MM. Hg | | | | | | | | | | | | |
| LEFT ATRIUM (MEAN) | LEFT VENTRICLE S/D | AORTA S/D (MEAN) | BRACHIAL ARTERY S/D (MEAN) | LEFT VENTRICULAR EJECTION (MEAN) | SYSTOLIC EJECTION PERIOD (SEC./BEAT) | LV—BA GRADIENT (MM. Hg)* | AORTIC VALVE FLOW (C.C./S.E.P. SEC.) | AORTIC VALVE AREA (CM.²) | LEFT VENTRICULAR WORK, EFFECTIVE (KG./M./MIN.) | LEFT VENTRICULAR WORK, TOTAL (KG./M./MIN.) | LEFT VENTRICULAR STROKE WORK (GM./M./MIN.) |
| 18 | 214/24 | 100/90 (95) | 104/62 (80) | 143 | .28 | 75 | 148 | 0.4 | 2.85 | 5.14 | 54 |
| 12 | 200/16 | – | 120/80 (90) | 166 | .16 | 86 | 147 | 0.4 | 1.93 | 3.57 | 35 |
| 8 | 230/10 | 180/120 (130) | 180/120 (130) | 190 | .20 | 40 | 317 | 1.1 | 5.60 | 8.20 | 91 |
| 20 | 180/22 | 120/70 (90) | 130/70 (100) | 176 | .22 | 30 | 255 | 1.1 | 4.28 | 7.52 | 75 |
| 6 | 170/10 | 80/60 (70) | 100/60 (80) | 150 | .28 | 59 | 186 | 0.6 | 3.21 | 6.02 | 60 |
| 8 | 150/6 | 100/60 (80) | 110/60 (80) | 140 | .24 | 35 | 243 | 0.9 | 3.66 | 6.40 | 65 |
| 16 | 190/18 | 104/78 (86) | 106/70 (80) | 180 | .38 | 60 | 105 | 0.3 | 2.42 | 5.42 | 57 |
| 8 | 140/10 | – | 100/60 (80) | 116 | .26 | 31 | 179 | 0.7 | 2.87 | 4.15 | 43 |

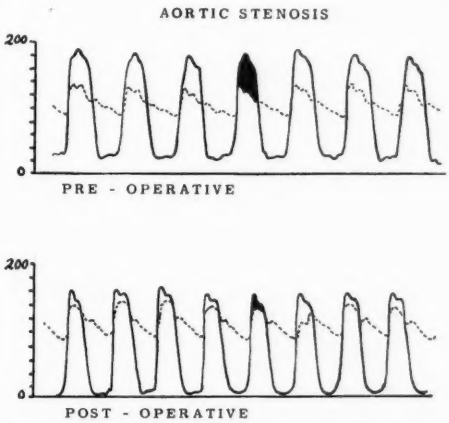
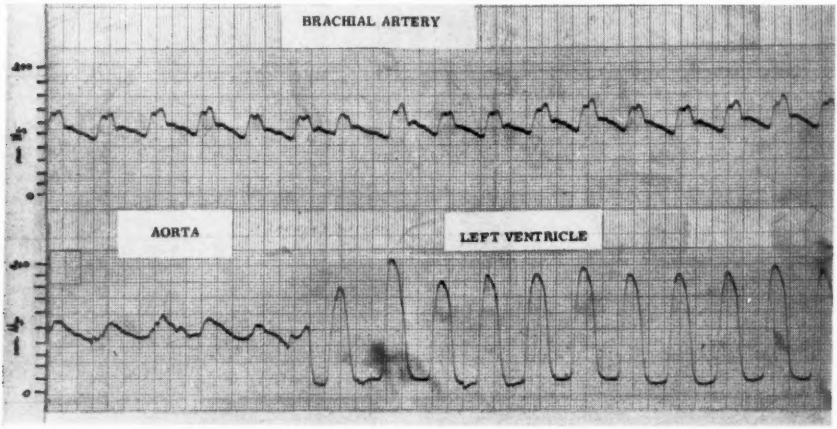
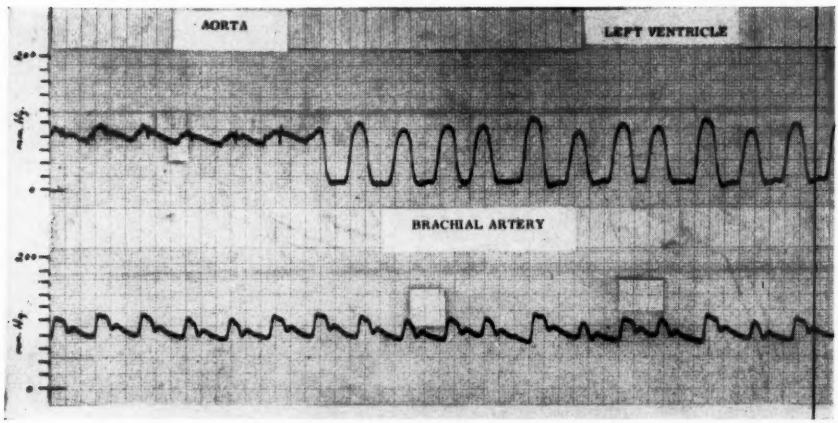



Fig. 2.—Superimposed brachial artery and left ventricular pressure tracings before and after aortic commissurotomy showing decrease in pressure gradient (shaded area).

The aortic valve flow of the group averaged 164 c.c./S.E.P. sec. with a range of 100 to 317 c.c. Normal in our laboratory is considered to be 175 to 250 c.c./S.E.P. sec.[18] Postoperatively the aortic valve flow increased in 9 patients, remained essentially the same in 2, and was decreased in 2. Of the 9 cases showing an increase, the range was 10 to 74 c.c. with an average increase of




*A.*




*B.*

Fig. 3.—Continuous pressure tracing recording from aorta to left ventricle before (*A*) and after (*B*) aortic commissurotomy (M. M.).

43 c.c./S.E.P. sec. Of the 2 patients in whom no change was noted, one had a subaortic lesion which was not improved by surgery, the other showed complete loss of pressure gradient and this patient had a cardiac output within

normal limits preoperatively. The 2 patients who showed a decrease in aortic valve flow were considered to have been in cardiac failure at the time of the postoperative catheterization.

The average cardiac index in the series before surgery was 2.3 L./min./M.$^2$ ranging from 1.5 to 3.1 L./min./M.$^2$ (normal range 2.6 to 3.6 L./min./M.$^2$). Postoperatively the cardiac index increased in 6 patients, remained unchanged in 4, and was decreased in 3 patients. Of the 6 cases showing an increase, 3 had a corresponding increase in the oxygen consumption.

Preoperatively the arteriovenous oxygen difference was elevated above normal in 3 patients and borderline in another 2 patients, indicating a blood flow inadequate fully to meet the tissue oxygen demands.

Postoperatively, the stroke volume was increased in 7 patients and decreased in 6. The average stroke volume preoperatively was 42 c.c. with a range of 25 to 62 c.c. Postoperatively the average for the group of 14 was unchanged (43 c.c.).

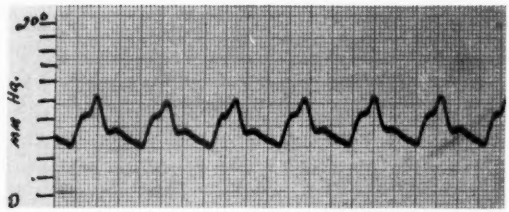
The left ventricular mean systolic ejection pressure was generally elevated preoperatively averaging 168 mm. Hg with a range of 140 to 193 mm. Hg. (Normal range: 70 to 126 mm. Hg.) A decrease occurred in 11 patients following surgery. In 3 patients a slight increase developed. In a fourth case (J. S.), the value remained unchanged.

A marked fall in the left ventricular systolic pressure was noted. It dropped from an average of 204 mm. Hg preoperatively to 162 mm. Hg postoperatively. A fall in this value occurred in every patient of the series.

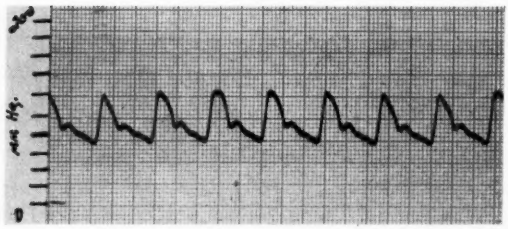
The left ventricular end-diastolic pressure tended to be elevated preoperatively with an average value of 18 mm. Hg and a range from 7 to 26 mm. Hg. The normal value is considered to be less than 10 mm. Hg. Postoperatively there was very little change, the average value being 15 mm. Hg. In 10 patients there was a very slight decrease, in 3 an increase was noted, and in one patient the end-diastolic pressure remained unchanged.

Preoperatively the left atrial mean pressure averaged 14 mm. Hg with a range of 6 to 27 mm. Hg. The pressure tracing showed giant A waves in patients with normal sinus rhythm typical of aortic stenosis. They did not show a consistent change in size or pattern as a result of aortic valve surgery. No change was noted in the average left atrial mean pressure postoperatively.

The brachial artery mean pressure averaged 95 mm. Hg preoperatively with a range of 80 to 130 mm. Hg. Instead of an expected rise postoperatively, a slight decrease was noted, the average being 88 mm. Hg with a range of 70 to 100 mm. Hg. Similarly the brachial arterial pulse pressure, which averaged 46 mm. Hg, showed essentially no change after surgery. The mean central aortic pressure values paralleled those of the brachial artery. The brachial artery pressure tracings preoperatively demonstrated the abnormalities previously described.[22] The most common finding was the presence of a notch or slur on the upstroke, with a delay of the peak of systole. Double peaked tracings or a notch on the downstroke between the peak of the curve and the normal dicrotic incisura (due to closure of the aortic valve) also were

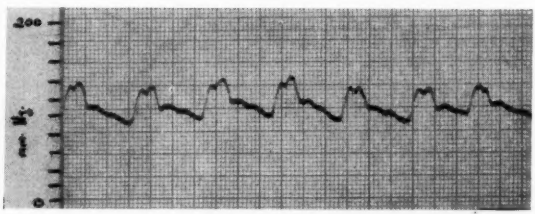



*A.*

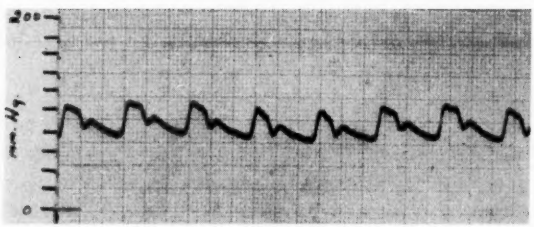



*B.*

Fig. 4.—Brachial artery pressure tracings pre- and postoperatively, showing disappearance of the anacrotic notch following surgery. There were small changes in gradient, flow, and area in this case (S. F.). *A*, Preoperative. *B*, Postoperative.




*A.*




*B.*

Fig. 5.—Brachial artery tracings pre- and postoperatively, showing loss of the anacrotic notch following surgery. The pressure gradient was abolished in this case (M. M.). *A*, Preoperative. *B*, Postoperative.

observed. The changes in these tracings following surgery were not always consistent with the changes in aortic pressure gradient, flow, or area. For example, the anacrotic notch may disappear postoperatively yet, the gradient, flow, and aortic area change little (Fig. 4). Significant changes in the pressure gradient flow may not be reflected by the brachial artery tracing. In only one case, the brachial artery tracing became more normal in appearance with the abolition of the pressure gradient (Fig. 5). There was generally little change in the pulmonary circuit following surgery. Pulmonary hypertension, to some degree, persisted in those cases presenting this finding preoperatively.

## DISCUSSION

Relief of the significant pressure gradient which exists across the aortic valve in aortic stenosis is dependent upon functional relief of the obstruction at the valve. Even after satisfactory mechanical mobilization of the fused valve elements there remains resistance to flow due to stiffened, calcified valve leaflets leaving a residual element of functional stenosis. This may account for the residual pressure gradient which is observed in the majority of patients post-commissurotomy. Another factor not infrequently responsible for a residual gradient is the inability of the surgeon to open all 3 commissures completely due to the presence of calcific deposits and inherent restrictions of the purely instrumental transventricular and the digitally guided transaortic operative techniques to which we have been limited in the past. The recent application of the open technique may prove advantageous in these respects.[26, 27] Subaortic stenosis is a lesion which is not satisfactorily relieved by the older techniques, but may be treated adequately by open heart surgery. The valvular morphology in the 2 cases in which the gradient was completely relieved did not include any calcification detectable either by x-ray or by digital examination at the time of surgery.

In a physiologic study of 37 cases of aortic stenosis by simultaneous (combined) left and right heart catheterization,[23] it was observed that 80 per cent had calculated valve areas less than 0.6 cm.$^2$ This supports the work of Gorlin and associates[24] and is further evidence that a critical functional valve area exists at about this point. While the estimated valve area postoperatively did not closely approach a normal area, except possibly in the 2 patients in whom the pressure gradient was abolished by surgery, the average calculated size of 0.8 cm.$^2$ probably explains the relief of the major symptom triad, angina, syncope, and dyspnea observed after aortic commissurotomy.[25]

With a fixed aortic valve orifice, the pressure gradient varies as the square of the flow. Hence, as valve areas are increased with little change in pressure gradient, there may be large changes in flow.

The high left ventricular end-diastolic pressure values in aortic stenosis probably are due to (1) left ventricular failure, and (2) alteration in the pressure volume relationships within the left ventricle secondary to concentric hypertrophy which is characteristic of the disease. It would follow,

therefore, that little or no change would be noted in this value in the early postoperative period if the diastolic pressure elevation were due to this latter factor.

Related to this altered pressure volume curve of the left ventricle, and hence, increased resistance to filling of this chamber during diastole, is the failure of the left atrial pressure and left atrial A waves to diminish postoperatively. It is likely that giant A waves result from efforts of the left atrial contraction to overcome the elevated left ventricular end-diastolic pressure during ventricular filling.[16] Therefore, they are likely to persist unchanged as long as the left ventricular hypertrophy and high end-diastolic pressure exist. The pulmonary circuit is generally within normal limits in aortic stenosis. Pulmonary hypertension in aortic stenosis may be due to (1) left ventricular failure or (2) increased pulmonary resistance secondary to vascular changes.[22] Persistence of pulmonary hypertension in the absence of left ventricular failure, as observed in some patients catheterized six months postoperatively, would seem to substantiate the existence of vascular changes.

The postoperative physiologic data obtained from this series of 14 patients indicate clearly that markedly altered hemodynamics persist after the performance of aortic commissurotomy in most cases. The residual pathologic physiology is dependent upon 2 main factors: (1) the inability of existing surgical techniques completely to restore the aortic valve to normal function, and (2) the secondary pathologic changes which have taken place in the myocardium of the left ventricle and atrium as a result of long-term obstruction.

In most cases of this series only 2 commissures could be opened by the surgeon. In a few certain cases it was possible only to split one commissure. Open heart surgical techniques under direct vision, using a pump oxygenator[28, 29] and retrograde perfusion of the coronary sinus,[30] would seem to offer an ideal method for the conquest of these obstacles. However, even complete fracture of all commissures to the annulus does not ensure complete functional relief of the stenosis in a very stiffened or heavily calcified valve. Valve leaflets which are thick and stiff continue to offer an element of impedance to passage of blood and a pressure gradient must exist in order to maintain flow at a reasonable level. This functional stenosis has been demonstrated beautifully by MacMillan with the use of a cyclic perfusion apparatus.[31] Obviously actual replacements of these severely calcified valves would be necessary to return the dynamics to normality.

While postoperative physiologic studies revealed considerable deviation from the normal, clinical follow-up[25] shows considerable relief from the three major debilitating symptoms, angina, syncope, and dyspnea. Until open heart surgical methods are more completely perfected it is apparent that existing surgical techniques are capable of relieving the aortic obstruction at least beyond the critical level of 0.6 cm.$^2$ Therefore, surgery is indicated for the

relief of severe major symptoms when present. Judicious medical management is necessary after surgery because of the secondary pathologic changes which often exist in these severe cases.

A small proportion of individuals with aortic stenosis show fibrous obliteration of the commissures with little or no calcification. These are much better suited to aortic commissurotomy by existing closed surgical techniques and it follows that better clinical results will be obtained provided that advanced left ventricular hypertrophy and failure are not present.

The data presented in this study support the opinion of other work that aortic obstruction should be relieved before advanced valve calcification occurs and before serious irreversible left ventricular damage has taken place. The development of these changes must be prevented if completely satisfactory results are to be obtained.

## SUMMARY

1. Simultaneously (combined) left and right heart catheterization data is presented on 14 patients with aortic stenosis, before and after surgery.

2. The details of the effect of aortic commissurotomy on the pathologic physiology of this lesion are described.

3. The failure of the altered physiology to be restored completely to normal by aortic commissurotomy is pointed out and certain reasons for it are postulated.

4. While aortic commissurotomy fails to restore the circulatory hemodynamics to normal in the majority of individuals, the relief of the symptom triad, angina, syncope, and dyspnea is accomplished in many cases. The rationale for this relief is discussed.

5. Early surgical relief of aortic stenosis is suggested in order to avoid advanced secondary valvular deformation and irreversible myocardial damage.

6. It appears that with the recent developments of open heart surgery, direct vision technique not only may permit the performance of a much more adequate commissurotomy, but offers hope that replacement of the valve will some day be possible in severely calcified cases.

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# Announcements

THE AMERICAN ASSOCIATION FOR THORACIC SURGERY

## Notice of Annual Meeting

The Thirty-eighth Annual Meeting of the American Association for Thoracic Surgery will be held May 16, 17, and 18, 1958, in Boston, Massachusetts. Headquarters will be at the Hotel Statler.

## Requests for Hotel Reservations

These should be mailed directly to Mr. F. Whitehouse, Front Office Manager, Hotel Statler, Park Square, Boston, Massachusetts. Please mention this Association, the type of accommodation desired, the date, and approximate hour of arrival and departure. If accommodations are desired elsewhere in Boston, please communicate directly with the hotel of your choice.

## Thoracic Surgery Forum

The Thoracic Surgery Forum will be continued as a special session at the 1958 meeting. The Forum is primarily for the presentation of short papers on current thoracic research, experimental problems, anatomical studies, and modified or new surgical techniques. The inclusion of any considerable amount of clinical material in these reports makes them unsuited for this session.

Papers at this session will be limited to ten minutes, including the showing of lantern slides or other illustrative material. At the discretion of the President, three minutes may be allowed as the maximum discussion for each paper.

All Thoracic Surgery Forum papers will be published together in one issue of the JOURNAL. Maximum length: 3,000 words without illustrations. If illustrated, an appropriate number of words must be subtracted for each cut used.

## Abstracts of Papers

Abstracts of all papers for presentation at the 1958 meeting must be received on or before Dec. 15, 1957, otherwise they will not be considered by the Program Committee.

Abstracts should be labeled "For Thoracic Surgery Forum," or "For Regular Program." They should contain from 200 to 250 words and accurately reflect the content of the completed paper.

*Six, repeat, six* clearly legible copies of *each* abstract must be sent to Miss Ada Hanvey, Administrative Assistant, The American Association for Thoracic Surgery, 600 South Kingshighway, St. Louis 10, Missouri.

Essayists selected for the program are reminded that papers presented at the meeting *must* be handed to the secretary immediately after their presentation. If they are not ready at that time there is a chance that they will not be published in THE JOURNAL OF THORACIC SURGERY.

### Motion Picture Session

Provisions have been made to show a limited number of motion pictures to be selected on a competitive basis. *The films themselves,* together with descriptive material concerning length of presentation, etc., must be received by Dr. Karl P. Klassen, The Ohio State University, Columbus 10, Ohio, before Dec. 15, 1957.

It is planned to run the motion pictures in a smaller room at the same time as the scientific sessions. If the films are well labeled, it will not be necessary for the author to be present for comment.

### Applications for Membership

Applications for Associate Membership in the Association must be received by the Membership Committee not later than Dec. 15, 1957, otherwise the application will be deferred for consideration until the 1959 meeting.

Applicants must be sponsored by three Active or Senior Members of the Association. In addition to the signatures on the application form, the sponsors will forward a *separate* letter concerning the applicant directly to:

Dr. Duane Carr
Chairman of the Membership Committee
899 Madison Avenue
Memphis, Tennessee

Sponsors are reminded that *new* letters of recommendation must be forwarded to the Membership Committee should their applicants be held over for reconsideration a *second year.* The new letters of recommendation should also be sent to Dr. Carr, the Chairman of the Membership Committee.

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